This is a digital copy of a book that was preserved for generations on library shelves before it was carefully scanned by Google as part of a project to make the world's books discoverable online.

It has survived long enough for the copyright to expire and the book to enter the public domain. A public domain book is one that was never subject to copyright or whose legal copyright term has expired. Whether a book is in the public domain may vary country to country. Public domain books are our gateways to the past, representing a wealth of history, culture and knowledge that's often difficult to discover.

Marks, notations and other marginalia present in the original volume will appear in this file - a reminder of this book's long journey from the publisher to a library and finally to you.

Usage guidelines

Google is proud to partner with libraries to digitize public domain materials and make them widely accessible. Public domain books belong to the public and we are merely their custodians. Nevertheless, this work is expensive, so in order to keep providing this resource, we have taken steps to prevent abuse by commercial parties, including placing technical restrictions on automated querying.

We also ask that you:

+ *Make non-commercial use of the files* We designed Google Book Search for use by individuals, and we request that you use these files for personal, non-commercial purposes.

+ *Refrain from automated querying* Do not send automated queries of any sort to Google's system: If you are conducting research on machine translation, optical character recognition or other areas where access to a large amount of text is helpful, please contact us. We encourage the use of public domain materials for these purposes and may be able to help.

+ *Maintain attribution* The Google "watermark" you see on each file is essential for informing people about this project and helping them find additional materials through Google Book Search. Please do not remove it.

+ *Keep it legal* Whatever your use, remember that you are responsible for ensuring that what you are doing is legal. Do not assume that just because we believe a book is in the public domain for users in the United States, that the work is also in the public domain for users in other countries. Whether a book is still in copyright varies from country to country, and we can't offer guidance on whether any specific use of any specific book is allowed. Please do not assume that a book's appearance in Google Book Search means it can be used in any manner anywhere in the world. Copyright infringement liability can be quite severe.

About Google Book Search

Google's mission is to organize the world's information and to make it universally accessible and useful. Google Book Search helps readers discover the world's books while helping authors and publishers reach new audiences. You can search through the full text of this book on the web at `http://books.google.com/`

LANE MEDICAL LIBRARY STANFORD
L756 .F786 1898 STOR
The diseases of the lungs / by James Kin
24503426331

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885
HENRY LEE DODGE MEMORIAL

LANE

MEDICAL LIBRARY

SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
SEMPER VIRENS
1885

HENRY LEE DODGE MEMORIAL

LANE
MEDICAL LIBRARY
SEAL OF THE LELAND STANFORD JUNIOR UNIVERSITY
1885
HENRY LEE DODGE MEMORIAL

THE DISEASES OF THE LUNGS

THE

DISEASES OF THE LUNGS

BY

JAMES KINGSTON FOWLER, M.A., M.D., F.R.C.P.
PHYSICIAN TO THE MIDDLESEX HOSPITAL AND TO THE
HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST, BROMPTON
LATE EXAMINER IN MEDICINE AT THE UNIVERSITY OF CAMBRIDGE AND ON THE
CONJOINT EXAMINING BOARD IN ENGLAND

AND

RICKMAN JOHN GODLEE, M.S., F.R.C.S.
FELLOW AND PROFESSOR OF CLINICAL SURGERY, UNIVERSITY COLLEGE, LONDON
SURGEON TO UNIVERSITY COLLEGE HOSPITAL AND TO THE HOSPITAL FOR CONSUMPTION
AND DISEASES OF THE CHEST, BROMPTON
SURGEON IN ORDINARY TO HER MAJESTY'S HOUSEHOLD

WITH ONE HUNDRED AND SIXTY ILLUSTRATIONS

LONGMANS, GREEN, AND CO.
39 PATERNOSTER ROW, LONDON
NEW YORK AND BOMBAY
1898

All rights reserved

THE

DISEASES OF THE LUNGS

BY

JAMES KINGSTON FOWLER, M.A., M.D., F.R.C.P.

PHYSICIAN TO THE MIDDLESEX HOSPITAL AND TO THE
HOSPITAL FOR CONSUMPTION AND DISEASES OF THE CHEST, BROMPTON
LATE EXAMINER IN MEDICINE AT THE UNIVERSITY OF CAMBRIDGE AND ON THE
CONJOINT EXAMINING BOARD IN ENGLAND

AND

RICKMAN JOHN GODLEE, M.S., F.R.C.S.

FELLOW AND PROFESSOR OF CLINICAL SURGERY, UNIVERSITY COLLEGE, LONDON
SURGEON TO UNIVERSITY COLLEGE HOSPITAL AND TO THE HOSPITAL FOR CONSUMPTION
AND DISEASES OF THE CHEST, BROMPTON
SURGEON IN ORDINARY TO HER MAJESTY'S HOUSEHOLD

WITH ONE HUNDRED AND SIXTY ILLUSTRATIONS

LONGMANS, GREEN, AND CO.
39 PATERNOSTER ROW, LONDON
NEW YORK AND BOMBAY
1898

All rights reserved

L756
F786
1898

PREFACE

In this work an endeavour has been made, by the association in authorship of a physician and a surgeon, to present a continuous picture of the medical and surgical aspects of pulmonary disease.

The book does not aspire to the position of a treatise on the diseases of the lungs, the object kept in view throughout having been to produce a work likely to prove useful to those engaged in the practice of medicine.

The authors have read and discussed each other's work, but it has been thought advisable in order to obviate any doubt which might arise as to the identity of the writer who expresses an opinion or recommends any particular method of treatment, that each chapter should be initialled by the author who is responsible for it.

An anatomical chapter has been introduced, which, while not intended to include all that can be said about the anatomy of the lungs and the surrounding parts, will, it is believed, be found to contain, in an accessible form, a number of useful facts such as the reader would only be able to discover with much labour in the textbooks of anatomy. In the revision of this chapter they have had the invaluable help of Professor G. D. THANE, of University College.

It is hoped that due acknowledgment has been made in the text of the indebtedness of the authors to the works of previous writers, and particularly, as regards the medical aspect of the subject, to the masterly treatise of Dr. WILSON FOX on 'Diseases of the Lungs and Pleura.'

28801

The valuable material contained in the clinical and pathological records of the Hospital for Consumption and Diseases of the Chest, Brompton, has supplied the authors with many illustrative cases, and they desire to thank their colleagues there for permission to make use of the notes.

The thanks of the medical author are especially due to his friends Dr. PERCY KIDD and Dr. A. F. VOELCKER, for the loan of microscopical specimens and for other assistance, and of both authors to Dr. J. J. PERKINS, for his kindness in undertaking to read the proofs and prepare the Index.

J. K. FOWLER.

R. J. GODLEE.

March 1898.

CONTENTS

CHAPTER		PAGE
I.	ANATOMICAL	1
II.	PHYSICAL DIAGNOSIS	50
III.	DISEASES OF THE TRACHEA	71
IV.	ACUTE BRONCHITIS	87
V.	CHRONIC BRONCHITIS	103
VI.	SECONDARY BRONCHITIS	115
VII.	PLASTIC BRONCHITIS	119
VIII.	BRONCHIECTASIS	123
IX.	BRONCHIECTASIS IN CHILDREN	143
X.	BRONCHIAL STENOSIS AND OBSTRUCTION	150
XI.	DISEASES OF THE BRONCHIAL GLANDS	153
XII.	EMPHYSEMA OF THE LUNGS	158
XIII.	INTERLOBULAR OR INTERSTITIAL EMPHYSEMA	181
XIV.	ASTHMA	183
XV.	PNEUMONIA	195
XVI.	PNEUMONIA (*continued*)	215
XVII.	PNEUMONIA (*continued*)	226
XVIII.	PNEUMONIA TERMINATING IN FIBROSIS (SUB-ACUTE INDURATIVE PNEUMONIA)	236
XIX.	ABSCESS OF THE LUNG	241
XX.	GANGRENE OF THE LUNGS	244

LIST OF ILLUSTRATIONS

PLATES

PLATE		TO FACE PAGE
I.	(Fig. 1) Actinomycosis of the Liver, bursting through the Ribs, and (Fig. 2) of the Lung and Pleura (coloured) (*vide* also Figs. 114 and 119)	456
II.	Part of the Liver shown in Plate I. Fig. 1 (*vide* also Fig. 119)	456
III.	Cells found in clear Inflammatory Pleuritic Effusions	552
IV.	Photograph of hand showing Clubbing of the Fingers from a case of Bronchiectasis	706
V.	Skiagram of the same hand, showing absence of enlargement of the Bones	707

ILLUSTRATIONS IN THE TEXT

FIG.		PAGE
1.	Frontal Section through the Trunk	2
2.	Sagittal Section through the Trunk (Right)	3
3.	Sagittal Section through the Trunk (Left)	3
4.	Sagittal Section through the Trunk (Median)	4
5.	Diagram of the Fifth Thoracic Vertebra and Costal Arch	5
6.	Diagram of the First and Seventh Ribs	5
7.	Interchondral Articulations	7
8.	Diaphragm of Medium Height in the Relaxed and in the Contracted Position	9
9, 10.	Highly Arched Diaphragm from the Front and the Side in the Relaxed and in the Contracted Conditions	10
11.	The Diaphragm seen from Above	11
12.	The Diaphragm seen from Behind	11
13.	Normal Male Chest	12
14.	Normal Female Chest	13
15.	Infant Chest	14
16.	Emphysematous Chest	15

FIG.		PAGE
17.	Phthinoid Chest	15
18.	Phthinoid Chest	15
19.	Section through Third Dorsal Vertebra	19
20.	Section through Fourth Dorsal Vertebra	20
21.	Section through Sixth Dorsal Vertebra	20
22.	Section through Eighth Dorsal Vertebra	21
23.	Section through Ninth Dorsal Vertebra	22
24.	Back View of Chest of an old man with Spine and Ribs removed	24
25.	Back View of the Sternum, showing the Relations of the Pleuræ to the Front of the Chest	25
26.	Appearances seen in the case from which the Sternum shown in Fig. 25 was removed	26
27.	Back View of the Sternum, showing the less usual arrangement of the Anterior Border of the Left Pleura	27
28.	Front of Chest, showing relations of Lungs and Pleura	28
29.	Back View of Chest, showing relations of Lungs and Pleura	29
30.	View of Right Side of Chest	30
31.	View of Left Side of Chest	31
32.	Inner Aspect of Right Lung (from His's Model)	32
33.	Inner Aspect of Left Lung (from His's Model)	33
34.	Outer Aspect of Right Lung (from His's Model)	34
35.	Outer Aspect of Left Lung (from His's Model)	35
36.	Outer Aspect of Chest	36
37.	Root of the Lung and the Mediastinum	37
38.	View of the Arrangement of the Structures and Organs on the Left Side	38
39.	The same as Fig. 38, after Removal of the Lung showing the root of the Lung	39
40.	Cast of the Interior of the Trachea and Bronchi (Aeby)	43
41.	Cast of the Interior of the Trachea and Bronchi (Aeby)	44
42.	The Lower End of the Trachea, its Divisions into the two Bronchial Trunks and the Course and Chief Divisions of these within the Lungs from Before (after Aeby)	45
43.	Dissection of the Bronchi and Pulmonary Vessels from the Side	47
44.	Diphtheria of the Larynx, Trachea and Bronchi	73
45.	Tubercular Ulceration of the Trachea	75
46.	Syphilitic Stenosis of the Trachea and Main Bronchi	78
47.	Syphilitic Stenosis of the Trachea	79
48.	Stenosis of the Trachea from Enlargement of the Thyroid Gland	80
49.	A Growth containing Thyroid Gland Structure compressing the Trachea	81
50.	Section of the same Growth, and of the contained Tumour and Trachea, showing the degree of Stenosis	81
51.	Perforation of the Trachea by an Enlarged Gland, and Discharge of the Gland into the Trachea	84
52.	Perforation of the Trachea by an Aneurysm of the Innominate Artery	85
53.	Microscopical Appearances in Acute Bronchitis	88
54.	Microscopical Appearances in Chronic Bronchitis	104
55.	Casts of the Tubes from a case of Plastic Bronchitis	120

FIG. PAGE
56. Cylindrical Bronchiectasis 124
57. Saccular Bronchiectasis 126
58. Section of a 'Honeycomb' Lung 145
59. Section through a Minute Cavity in a case of Honeycomb Lung . 146
60. Emphysema of the Lung 164
61. Curschmann's Spirals 185
62. Pneumococci in the Sputum from a case of Pneumonia . . . 199
63. Pneumonia, Red Hepatisation, Low Power 203
64. Pneumonia, Red Hepatisation, High Power 203
65. Pneumonia, Grey Hepatisation 204
66. Temperature Chart from a Case of Pneumonia, Crisis on Seventh Day 212
67. Temperature Chart from a Case of Pneumonia, Crisis on Seventh Day 213
68. Temperature Chart from a Case of Pneumonia, showing Fall by Lysis 216
69. Temperature Chart from a Case of Pneumonia, showing Fall by Lysis and subsequent Recurrence of Pyrexia 217
70. Temperature Chart from a Case of Pneumonia of Prolonged Duration 218
71. Temperature Chart from a Case of Pneumonia, Sudden Fall of Temperature following the Application of an Ice-bag. Subsequent Recurrence of Fever 219
72. Temperature Chart from a Case of Pneumonia, Fatal on the Twelfth Day 220
73. Temperature Chart from a Case of Pneumonia, Fatal on the Seventh Day, showing moderate degree of Pyrexia 221
74. Curve of Mortality from Pneumonia at Different Age Periods . . 229
75. Sub-acute Indurative Pneumonia 239
76. Microscopical Appearances in Broncho-pneumonia 254
77. Pulmonary Fibrosis 262
78. Acute Pleurisy with Thickening of the Connective Tissue of the Lung 263
79. Reticular Fibrosis of the Lung with Emphysema 267
80. Congestion of the Lung 278
81. Œdema of the Lung 279
82. Hypostatic Pneumonia 280
83. Tubercle Bacilli in Sputum (coloured) 297
84. Tubercle 300
85. Tubercle undergoing Caseation 303
86. Tubercle undergoing Fibrosis 304
87. Continuous Type of Pyrexia in Miliary Tuberculosis 325
88. Inverse Type of Pyrexia in Miliary Tuberculosis 326
89. Inverse Type of Pyrexia in Miliary Tuberculosis 326
90. Pyrexia in Caseous Tuberculosis 327
91. Chart from a Case of Caseous Tuberculosis 327
92. Pyrexia in Caseous Tuberculosis, with Failure of Vital Power . 328
93. Chart from a Case of Fibroid Tuberculosis 328
94. Chart from a Case of Chronic Tuberculosis, Quiescent . . . 329
5. Chart from a Case of Chronic Tuberculosis, Infiltration with commencing Softening 329

FIG. PAGE
96. Diagram of a Vertical Median Section of the Lung, showing one of the Sites of Primary Infiltration in Tuberculosis, &c. 351
97. Diagram of Lateral Aspect of the Lung, showing a Less Usual Site of Primary Disease of the Apex 351
98. Diagram showing the Relations of the Lobes of the Lungs to the Chest Wall Posteriorly 352
99. Diagram illustrating the Extension of a Lesion of the Lower Lobe along the Interlobar Septum 353
100. Showing the Position of the Arm in which the Vertebral Border of the Scapula indicates (roughly) the Usual Line of Extension of Lesions along the Interlobar Septum 353
101. Showing the Usual Mode of Extension of Tubercular Disease towards the Base of the Lung 354
102. Typical Arrangement of Lesions in a Case of Apical Tuberculosis, with Non-tuberculous Disease of the Lower Lobe 354
103. Lateral View of Lung, showing an Occasional Site of Lesion in a Lung not primarily affected 355
104 & 105. Illustrating a 'Crossed Lesion' 355
106. Site of Lesions in Secondary Tubercular Infection of the Lung . . 356
107. Illustrating an Unusual Arrangement of Lesions in the Lower Lobe 356
108. Elastic Tissue of the Lung, from the Sputum of a Case of Tuberculosis 367
109. Pyrexia during the 'Reaction' produced by the Injection of Tuberculin 399
110. Chart showing the Effect upon the Temperature of the Administration of Phenacetin and of the omission of the Drug . . . 405
111. Forceps for the Extraction of a Foreign Body from a Bronchus . 426
112. Bent Wire for the Extraction of a Foreign Body from a Bronchus . 427
113. Changes in the Lung Secondary to Syphilitic Stenosis of the Trachea and Main Bronchi 442
114. Front View of a Boy suffering from Actinomycosis of the Right Lung and Pleura 454
115. Back View of the same Patient 455
116. Actinomycosis; Clubs as usually found in Cattle 456
117. Actinomycosis; Method of Spreading. Low Power 456
118. Actinomycosis; Threads as usually found in Man (Highly Magnified) 456
119. Front View of Patient suffering from Actinomycosis of the Liver, affecting secondarily the Right Lung and Pleura 457
120. Aspergillus Fumigatus 461
121. A Tubercular-looking Nodule in the Lung in a Case of Aspergillo-mycosis 462
122. Mycelium in the Sputum in a Case of Aspergillo-mycosis . . . 463
123. Section of Lung from a Case of Protozoic Infection, secondary to Disease of the Skin (Coloured) 471
124. Hydatid Cyst in the Lung 473
125. Aneurysms of the Pulmonary Artery in Cavities in the Lung . . 516
126. Ruptured Aneurysm of the Pulmonary Artery in a Cavity in the Lung 516

FIG. PAGE
127. Chart showing Initial Lowering of Temperature and subsequent Pyrexia accompanying an Attack of Hæmoptysis 522
128. Microscopical Appearances in Acute Pleurisy 552
129. Diagram illustrating the Exudation of Fluid within a Sac lined by Coagulated Fibrin 560
130. Diagram showing the Physical Signs in a Case of Long-standing Large Effusion in the Left Pleura (Front Aspect) 561
131. Diagram showing the Physical Signs in a Case of Long-standing Large Effusion in the Left Pleura (Posterior Aspect) . . . 562
132. Thickening of the Pleura secondary to Chronic Tuberculosis of the Lung 579
133. Rib six weeks after a part was removed 597
134. Regenerated Rib after a part had been removed 597
135. Regenerated Rib after a part had been removed 598
136. Bony Ring formed by Bars on each Side of a Drainage Tube . . 598
137. Volkmann's Periosteum Elevator 599
138. Blunt Periosteum Elevator 599
139. Blunt Forceps for cutting Ribs 599
140. Operation for Empyema. The Patient Lying on the Back . . 601
141. Operation for Empyema. The Patient Lying on the Side . . 602
142. Drainage Tube 603
143. Lateral Curvature following Empyema 608
144. Result of Estlander's Operation 612
145. Chart showing the Rapid Fall of Temperature on the Occurrence of Pneumothorax 634
146. Chart from a Case in which Pneumothorax occurred without Marked Symptoms 635
147 & 148. Groups of Cells from a Case of Tumour of the Pleura . . 663
149. Mediastinal Lympho-sarcoma 681
150. Fleshy Processes covered with Thick Skin from which Hairs are Growing. From a Dermoid Cyst of the Lung 696
151. A Similar Fleshy Process 697
152. Hands of a Patient suffering from a Pulmonary Cavity showing Bony Enlargement 703
153. Skiagram of Hands and Forearm of a Patient, showing Bony Enlargement 704
154. Hands of a Patient in whom there was no evidence of Bony Enlargement 705

THE DISEASES OF THE LUNGS

CHAPTER I

ANATOMICAL

	PAGE
Introductory	1
Thoracic spine	2
The ribs	3
Anomalies of ribs	7
Intercostal spaces	8
The sternum	8
Anomalies of the sternum	9
The diaphragm	10
Shape of the chest altered by position	13
Other circumstances modifying the shape of the chest	13
The intercostal vessels	16
The intercostal nerves	18
Lymphatics of the thoracic wall	18
The mediastinum	19
The pleura	21
The lungs	28
Movements of the lung	40
The trachea	40
The bronchi and pulmonary arteries	42
Pulmonary veins	46
Bronchial arteries and veins	48
Lymphatics of the lungs	48
Lymphatics of the pleura	49

Introductory.—The modern study of anatomy by means o frozen sections has emphasised the fact that a small part only o the chest is occupied by the lungs. The marked projection forwar of the spine and the height to which the diaphragm reaches accoun for this in great part, and one result is that neither the anatom nor the pathology of the lungs and pleura can be dealt with withou discussing those of the surrounding parts, to say nothing of thos of the structures occupying the mediastinum. This is well illustrated by the appearances seen in a coronal section of the ches (fig. 1).

The bony framework of the chest is made up of the thoraci spine, the ribs, and the sternum.

Thoracic spine.—The thoracic spine, composed of the twelve thoracic vertebræ, presents normally two curves: one with the convexity backwards is always present; the other, a lateral one, usually with the convexity to the right but occasionally to the left, is often so slight as to be scarcely perceptible. The thoracic is the least flexible region of the movable part of the vertebral column, the antero-posterior movements being limited by the small amount of intervertebral substance and the imbrication of the spines and laminæ, the lateral movements by the approximation of the ribs; a slight degree of rotation about a vertical axis is, however, permitted. It is probably due to the slight obliquity of the articular processes that whenever exaggeration of the normal lateral curve of the spine occurs, as in scoliosis or after empyema, a certain amount of rotation or torsion is invariably present. As a

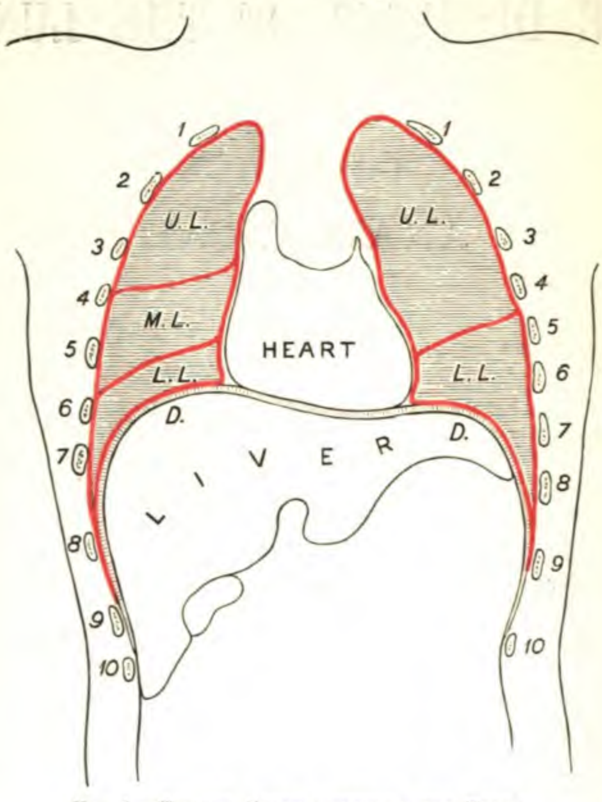


FIG. 1.—FRONTAL SECTION THROUGH THE TRUNK

From a plaster cast: 1 to 10, ribs; *U L*, *U L*, upper lobes of lungs; *M L*, middle lobe of right lung; *L L*, *L L*, lower lobes of lungs; *D*, diaphragm. The pleura is marked by a red line.

result of this torsion the spines always point towards the concavity of the curve, while the bodies of the vertebræ are twisted outwards, and consequently the angles of the ribs become more prominent on the convex side of the curve, and the rib cartilages on the concave side. These distortions of the chest modify very much the relations of the thoracic viscera to the surface of the body.

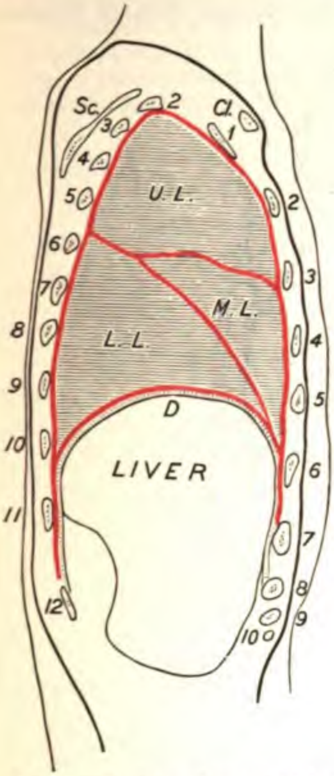


FIG. 2.—SAGITTAL SECTION (right). (After Rüdinger)

1 to 12, ribs; *Cl*, clavicle; *Sc*, scapula; *D*, diaphragm; *U L*, upper lobe of lung; *M L*, middle lobe of lung; *L L*, lower lobe of lung. The pleura is marked by a red line.

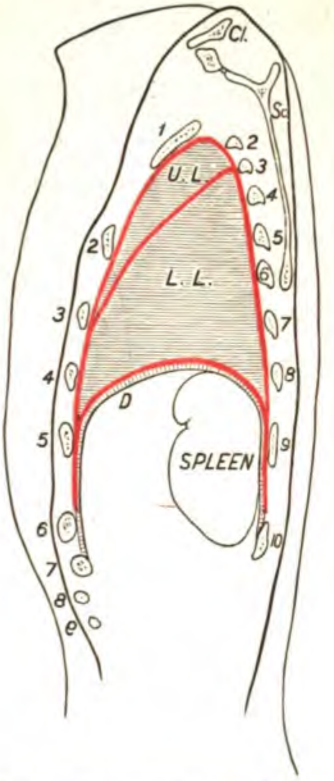


FIG. 3.—SAGITTAL SECTION (left). (After Rüdinger)

1 to 10, ribs; *Cl*, clavicle; *Sc*, scapula; *D*, diaphragm; *U L*, upper lobe of lung; *L L*, lower lobe of lung. The pleura is marked by a red line.

The manner in which the spine projects into the thoracic cavity, and forms as it were part of a septum dividing it into two parts, is dealt with in the next section.

The ribs.—The ribs, the shape of which approximates above to that of segments of circles, but which become sickle-shaped in the middle of the series and much straighter below, are attached to the spine with varying degrees of obliquity; but they all slope downwards, outwards, and forwards. They thus, together with their cartilages, the sternum, and the spine, form a hollow cone, open both above and below, flattened from before backwards, and extending further, both upwards and downwards, behind than in front. The enclosed space is very much encroached upon by the spine, so that there is a suggestion of a division into two laterally applied hollow cones, each of which is more or less circular, or rather oval, in horizontal section. The average distance between the sternum and the spine is not much more than about two-thirds of that between the angles of the ribs and front of the chest (figs. 2, 3, 4).

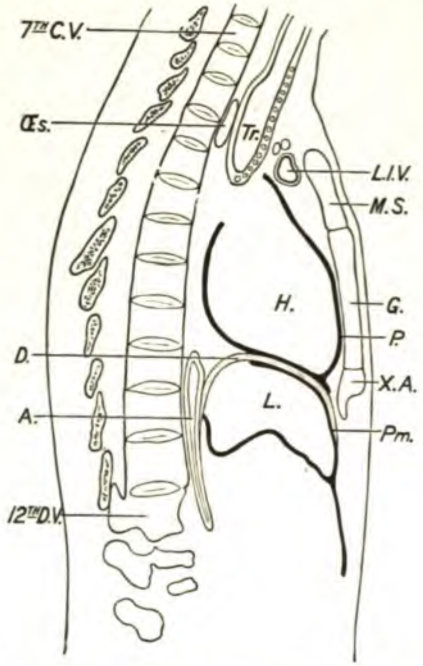


FIG. 4.—SAGITTAL SECTION (median). (After Rüdinger)

7th *C V*, seventh cervical vertebra; 12th *D V*, twelfth dorsal vertebra; *M S*, manubrium sterni; *G*, gladiolus; *X A*, xiphoid appendix; *Œs*, œsophagus; *Tr*, trachea; *L I V*, left innominate vein; *H*, heart; *P*, pericardium; *D*, diaphragm; *Pm*, peritoneum; *L*, liver; *A*, descending aorta. There was some lateral dorsal curve of the spine to the right.

The ribs are attached by synovial joints to the bodies and the transverse processes of the vertebræ, and in front they are directly continuous with their cartilages (except in the case of the first, where a synovial cavity is occasionally found). Between the cartilages of the true ribs and the sternum there are synovial joints, except in the case of the first and often of the sixth and seventh.

The joint of the second is commonly divided into two by a short inter-articular ligament.

The movement of the ribs is principally one of rotation upwards and downwards, round an axis, which is directed obliquely outwards and backwards, as well as somewhat downwards, passing through the costo-central articulation and the neck of the rib and a little in front of the costo-transverse joint (fig. 5).

As the ribs are directed obliquely downwards and forwards from the spine, the rotation upwards is necessarily accompanied by an increase of the sagittal (antero-posterior) diameter of the thorax (fig. 6), and as the axis of rotation is directed backwards and downwards the movement is also accompanied by an increase of the transverse diameter (fig. 5). The necks of the ribs are more oblique, and the axis of rotation is directed more backwards in the lower than in the upper part of the chest, and consequently the lateral expansion is greater below than above. The last two or three ribs move chiefly backwards in inspiration and forwards in expiration. Elevation of the first rib takes place to a considerable extent, but lateral expansion of this region is very limited. These movements could not, of course, take place were it not for the flexibility of the rib cartilages and the joints between them and the sternum; and the slight movement which is possible between the manubrium and the gladiolus no doubt aids them. Those rib cartilages are longer and

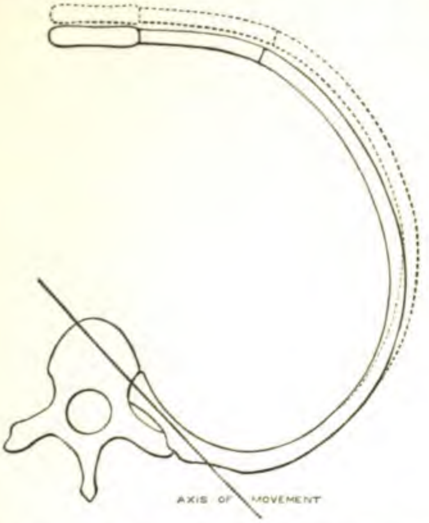


Fig. 5.—Diagram of the Fifth Thoracic Vertebra and Costal Arch

Projected into a horizontal plane, to show the axis of movement of the rib, and the manner in which the sagittal and coronal diameters of the thorax are increased. The continuous lines indicate the position in expiration, the broken lines in inspiration. (Professor G. D. Thane, from 'Quain's Anatomy.')

therefore more flexible, which correspond to the ribs whose excursions are greatest (seventh and eighth). When they become ossified from old age,[1] the expansion of the chest is limited as completely as when the rotation of the spinal end is prevented by caries of the spine or spondylitis deformans.

The line of the costo-chondral junctions on each side is, on the whole, directed downwards and outwards; that of the first rib is in an adult about 1½ inch from the sternum,[2] that of the second rib somewhat nearer the middle line; the others lie behind an oblique line drawn downwards and outwards from the latter point to the tip of the tenth rib.

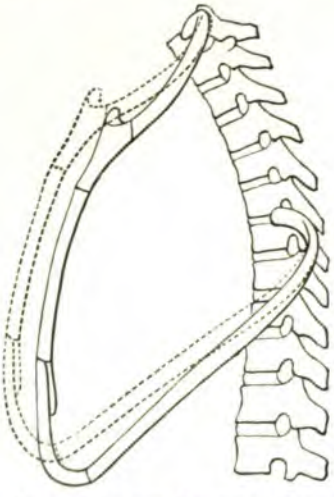


FIG. 6.—DIAGRAM OF THE FIRST AND SEVENTH RIBS

In connection with the spine and the sternum, showing how the latter is carried upwards and forwards in inspiration. The expiratory position is indicated by continuous lines, the inspiratory by broken lines. (Professor G. D. Thane, from 'Quain's Anatomy.')

The narrowest parts of the ribs are about a hand's breadth from the sternum, and the broadest parts a little outside the angles. The periosteum covering the ribs is strong; it is easily detachable from the outer surface, but is firmly fixed to the internal surface owing to the attachment of the intercostal muscles.

---

[1] This process consists in a deposit of bone round the costal cartilages, not a calcification or ossification of the whole of the cartilage. It is usually present to a greater or less extent round the cartilage of the first rib in adult males, sometimes forming a complete sheath round the cartilage. In advanced life the other cartilages become usually affected in the same way; but the degree to which it advances and the time at which it begins are liable to great variation. It is much less marked in women than in men, and in some quite old people the cartilages are remarkably free from bony deposit.

[2] See p. 8 with regard to the breadth of the sternum.

Anomalies of ribs.—Anomalies in the number and shape of the ribs are occasionally met with. A cervical rib sometimes forms a tumour in the supraclavicular region of the neck, but has no influence on the anatomy of the thorax. The twelfth rib varies in length from less than an inch to eight inches, and in many cases does not project beyond the edge of the erector spinæ. This may lead to confusion in counting the ribs if this be begun from below. A similar mistake may arise in the rare event of the presence of a lumbar rib of considerable length. Bifurcation of the anterior extremity of a rib and the corresponding cartilage is occasionally met with; or there may be an additional cartilage projecting from the side of the sternum. These anomalies are mostly unilateral, and the additional cartilage is almost always in the third interspace (fig. 7). Extremely prominent ribs or rib cartilages are not unfrequently met with, and, as far as I have seen, mostly in the same situation; these may give rise to the suspicion of tumour, and therefore claim a brief notice in this work. I have notes of at least three cases, of young women, to whom considerable mental anxiety, and apparently a good deal of pain, were caused by this anatomical peculiarity.

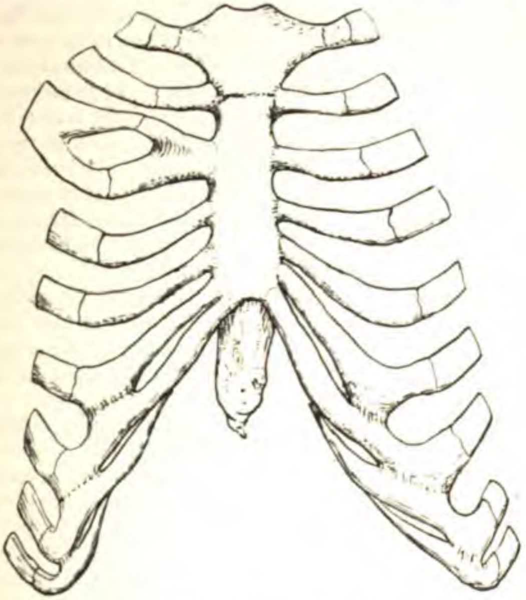


FIG. 7.—INTERCHONDRAL ARTICULATIONS

From a dissection by Professor G. D. Thane. The interchondral articulations are seen in the sixth, seventh and eighth interspaces. The third rib presents the common irregularity described on p. 6.

It is to be observed that the second cartilage is normally more prominent than the others, and prolongs horizontally outwards, the ridge formed by the sternal angle (see p. 8). The eighth rib is often a true rib—that is, it articulates directly with the sternum.

Intercostal spaces.—The intercostal spaces, filled by the intercostal muscles and aponeuroses, and containing the intercostal vessels and nerves, vary in width at different parts of the chest, and at different periods of the respiratory act. Modifications of the shape of the chest also cause great alterations in the distance between the ribs.

The second, third, tenth, and eleventh interspaces are, in a well-formed chest, the widest. The widest part of any individual space is near the anterior part of the rib (not between the cartilages). The anterior ends of the intercostal spaces tend to become pointed. This is only slightly marked in the first, third, and fourth interspaces, and not at all in the second. It is very obvious in the sixth, seventh, eighth, and ninth interspaces, some or all of which are completely interrupted by the interchondral articulations (fig. 7).

The assertion that the intercostal spaces become wider during inspiration is not, as regards them all, strictly true; though the fact that the ribs become more nearly horizontal than during expiration would, at first sight, seem to imply that this is the case. It is too complicated a subject for discussion in this work.

Extension of the spine widens the intercostal spaces, flexion of it contracts them, lateral bending of the spine widens those on one side and contracts those upon the other.

The intercostal spaces can be everywhere traced with the finger except where they are covered by the clavicle and the scapula and by the thick spinal muscles. It is important to recognise their normal condition in order to compare it with that of the undue prominence caused by intra-pleural effusions.

The sternum.—The sternum varies much in size, shape, and direction. It is usually longer in men than in women, and the gladiolus is, as a rule, relatively shorter in women than in men. The length of the sternum is not by any means always proportionate to the size of the individual; it varies not only in length, but also in breadth and thickness.

The general direction of the bone is commonly at an angle of twenty degrees to twenty-five degrees with the perpendicular, but the direction of the manubrium is not the same as that of the gladiolus. This gives rise to the sternal angle (*angulus Ludovici*) named after Louis of Paris.

Much discussion has taken place as to the importance of this angle in the movements of the chest, and as to the causes of its exaggeration in some cases. The reader is referred to an exhaustive article by the late Wilhelm Braune in the 'Archiv für Anatomie und Entwicklungsgeschichte,' 1888 (p. 304). Braune begins by stating that this angle has been used as the diagnostic sign of commencing phthisis, it being supposed that the contraction of the apices of the lungs causes a falling backwards of the manubrium. He says, however, that experience shows that exaggeration of the angle by no means always accompanies phthisis, but, on the other hand, is often met with when the lungs are normal, and very frequently in cases of emphysema. This angle is rendered more marked by exaggerated movements of inspiration, and if the manubrium be separated from the gladiolus, forcible inflation of the lungs makes it more prominent than before. He does not deny that, in very marked contraction of the apices, the manubrium may fall backwards.

By means of a simple mechanical contrivance it is easy to satisfy oneself of the actual size of this angle, and of the extent to which it is altered by the movements of respiration, it will be found to vary from about 170 degrees to 160 degrees, but it may be much more acute, and the difference in size caused by a deep inspiration, which may be as great as ten degrees or even more in healthy young women, is much less in adult men, and does not exist when the two parts of the bone are synostosed as in the rigid chests of the aged, or after that not very uncommon disease, strumous caries of the joint between the manubrium and the gladiolus.

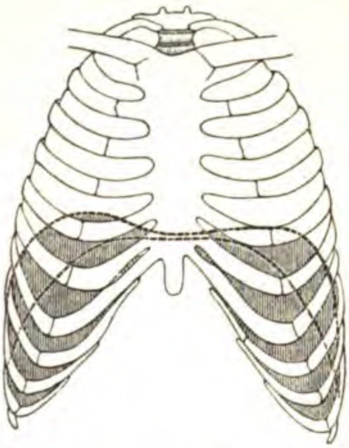


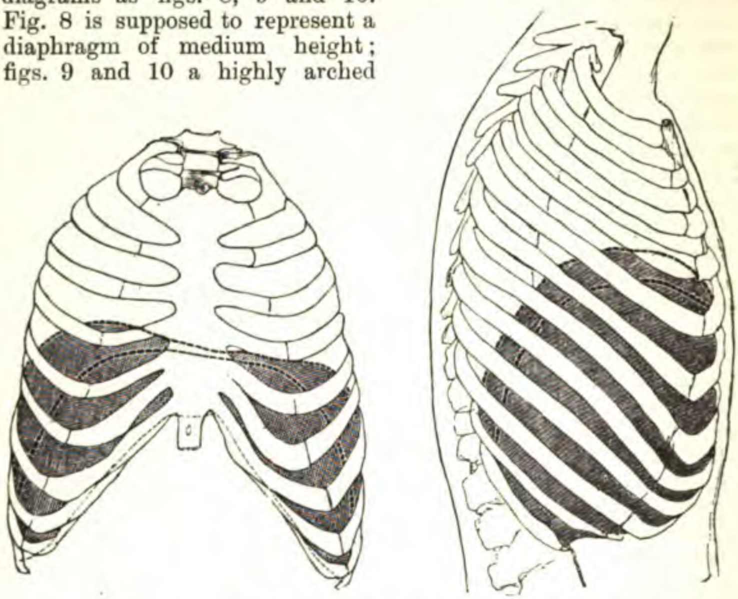
FIG. 8.—DIAPHRAGM OF MEDIUM HEIGHT IN THE RELAXED AND IN THE CONTRACTED POSITION (Pansch)

Anomalies of the sternum.—The sternum is liable to congenital defects, most of which are not of importance to the practical surgeon or physician. Passing over cases of ectopia cordis, mention must be made of deficiency of one half or the whole of the bone, with consequent uncovering of some part of the heart and the probable formation of a pulmonary hernia. Clefts in the sternum, which may involve complete non-union of the two halves, or may consist of triangular, oval, or elongated fissures, are occasionally met with. They are filled by strong membrane, and are not usually recognisable during life; but the existence of a cleft increases the danger of punctured wounds of the front of the chest. A small aperture (sternal foramen) in the lower part of the gladiolus is not uncommon. A deep hollow in the lower part of the sternal region is also occasionally met with congenitally, similar to that which is supposed to be produced by following such trades as that of a cobbler. Occasionally the manubrium is synostosed with the second segment of the bone, while the latter forms an articulation with the remainder of the gladiolus.

he jugular or suprasternal notch varies very much in breadth pth, owing to the direction of the surfaces on the manubrium for articulation with the clavicles and the distance between the two heads of the sterno-mastoid muscles.

The xiphoid appendix varies much in shape (it is often bifid), in length, and in its direction. Sometimes when it is directed very much backwards it appears to give rise to pain, and in that case may require removal. Often the seventh, and sometimes even the eighth, cartilages meet in front of it, under which circumstances it is scarcely to be felt through the abdominal walls.

Diaphragm.—The encroachment of the abdominal viscera on the space contained within the bony thorax is shown by such diagrams as figs. 8, 9 and 10. Fig. 8 is supposed to represent a diaphragm of medium height; figs. 9 and 10 a highly arched diaphragm such as may be met with in the young and powerful, and still more markedly in the newly born.



FIGS. 9 AND 10.—HIGHLY ARCHED DIAPHRAGM FROM THE FRONT AND THE SIDE IN THE RELAXED AND IN THE CONTRACTED CONDITION

Statements about the height of the diaphragm are open to the criticism that they result from post-mortem observations, in which the state of extreme expiration only is met with, and that consequently they may not apply to the condition existing during life. We are probably justified in stating that the part corresponding to the heart remains almost stationary during respiration, and that the vault or dome on the right side reaches in the state of rest to the level of the lower part of the fourth interspace in front, which corresponds in the scapular line to the eighth rib, and in the axillary line to the sixth rib. The left dome does not reach so high by one finger's breadth.

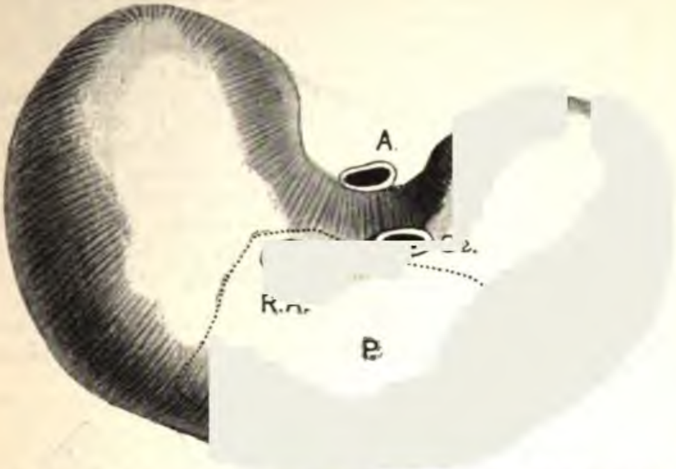


FIG. 11.—THE DIAPHRAGM SEEN FROM ABOVE, SHOWING THE PECULIAR SHAPE OF THE TENDON AND THE MANNER IN WHICH IT ADAPTS ITSELF TO THE SHAPE OF THE SPINE

A, aorta ; *Oe* œsophagus ; *R A*, right auricle ; *P*, pericardium.

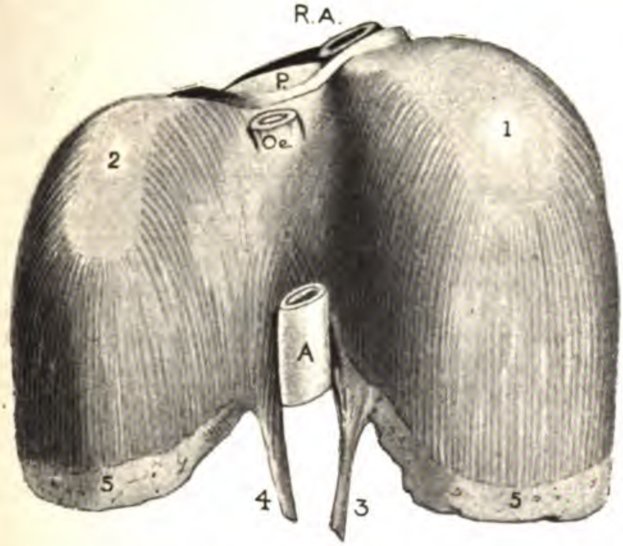


FIG. 12.—THE DIAPHRAGM SEEN FROM BEHIND

1, right side of tendon ; 2, left side of tendon ; 3, right crus ; 4, left crus ; 5, origin from ribs ; *A*, aorta ; *Oe*, œsophagus ; *P*, pericardium ; *R A*, right auricle.

Probably in cases of extreme arching it does not reach higher on the right side than the level of a line drawn through the third rib cartilages, while in cases of low arching the level will be that of the junction of the sixth rib cartilages with the sternum.

The shape of the diaphragm in a state of expiration may be gathered from the accompanying figures (figs. 11 and 12), drawn from a preparation of the muscle *in situ*, on a cast made by pouring plaster of Paris into the abdomen after the removal of the viscera. The great narrowing of the central part by the projection forward of the vertebral column, the inequality in the arching on the two sides, the irregular shape of the tendon and its very faint resemblance to that of a trefoil leaf, and the predominantly vertical direction of the muscular part are well shown.

It is impossible to define accurately the precise shape assumed by the diaphragm during extreme inspiration or when the pleura is distended. If the abdominal viscera be removed, and the lungs be inflated by blowing into a tube fitted into the trachea, the diaphragm can be made to project downwards below the level of the stationary part corresponding to the heart, and the descent of the liver under somewhat similar circumstances during life shows that a corresponding descent of the diaphragm has occurred. The removal of the abdominal viscera, however, so completely alters the physical conditions that too much stress must not be laid upon the details of such observations.

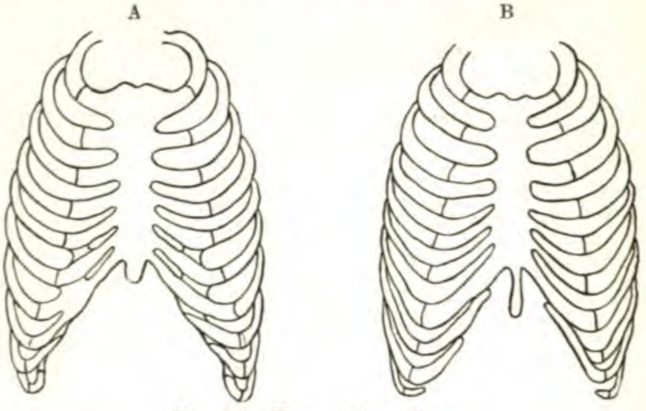


FIG. 13.—NORMAL MALE CHEST

A, æt. 28, from Luschka's 'Brustorgane des Menschen,' 1857; B, æt. about 50, from a preparation by the author.

The origin of the diaphragm and the central part of it being comparatively speaking fixed, it follows that the result of a contraction of its fibres will be a diminution in the height of the arch. But it must be remembered that the lower part of the thorax is by no means rigid, and in young people it is very flexible. If it be impossible to draw air into the lungs, as in cases of obstruction of the trachea, retraction of the lower part of the chest wall will naturally follow from the forcible contraction of the diaphragm. Hence arises the practice of observing the lower part of the chest in estimating the amount of obstruction to the upper air passages in cases of dyspnœa possibly requiring tracheotomy.

Under some circumstances, as, for example, when the chest has become very rigid, or when the intercostal muscles are paralysed, the diaphragm becomes the chief, or, indeed, the only muscle of respiration—respiration being then more or less completely abdominal. Under all circumstances it is a very important muscle, and any interference with its movements, as by the accumulation of food or gas in the stomach or fluid in the peritoneum, soon shows itself by causing dyspnœa.

The sternal portion of the diaphragm is not infrequently wanting, and in cases of diaphragmatic hernia much larger portions of the muscle may be absent.

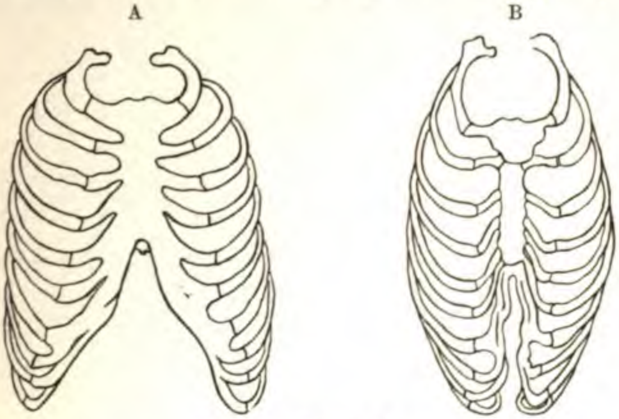


FIG. 14.—FEMALE CHEST

A, normal healthy subject, æt. 18, from Lucæ's 'Zur Anatomie des weiblichen Torso;' B, deformed by tight lacing, from Merkel's 'Handbuch der topographischen Anatomie.'

Shape of the chest altered by position.—The general shape of the chest is altered, like that of the abdomen, by changes of posture; for example, in the recumbent posture, the spine is straightened out and the upper ribs are somewhat raised, rendering further inspiratory movement difficult; and at the same time the abdominal viscera fall up against the diaphragm, tending to elevate it and opposing its descent. Hence arises the difficulty of breathing in the recumbent posture when dyspnœa is present.

Other circumstances modifying shape of the chest.—Something must be said about what may be called normal varieties of the shape of the chest, and other changes due to disease.

In the infant, and especially in the new-born child, the ribs are less inclined than in the adult, the thorax is short and conical owing to the straight spine, the small size of the lungs, and the large size of the liver (fig. 15).

In old age the forward curve of the spine becomes increased, and the lungs are probably atrophied, the ribs become more oblique, and the chest is altogether smaller, its movements also are much restricted if the cartilages are ossified.

The adult male chest is more or less conical, and the subcostal angle is broad (fig. 13).

The form of the adult female chest (fig. 14) differs from that of the male, not only in the relatively short sternum and delicacy of the ribs, but also in being more barrel-shaped—that is, less expanded below. Consequently the subcostal angle is smaller. The female chest is, in civilised countries, often much modified in shape by tight lacing. This, by constricting the lower ribs still further diminishes the subcostal angle; to such an extent indeed, sometimes, as to make some of the cartilages below the seventh touch or even overlap one another. This, of course, drives the liver downwards, and restricts the possible thoracic respiration to the upper part of the chest.

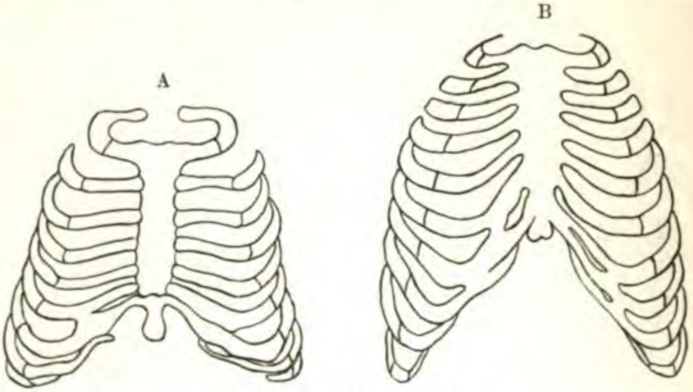


Fig. 15.—Infant Chest

A, at birth, from Henke's 'Anatomie des Kindesalters;' B, æt. 3 years, from a preparation by the author.

Prolonged decumbency may produce alteration in the curves of the spine, which depend upon the position habitually assumed by the patient. Those who are constantly propped up by pillows develop an exaggeration of the dorsal curve, while those who lie more flat lose some of the lumbar curve; indeed, this part of the spine may even become slightly convex backwards. Chronic bronchitis may also lead to an exaggeration of the dorsal curve.

In the emphysematous chest (fig. 16), the ribs approximate to the horizontal direction, the interspaces are widened and the subcostal angle is large, the thorax is said to be barrel-shaped, but it is really more nearly conical than in the normal condition. It may be said to correspond to a full inspiratory type.

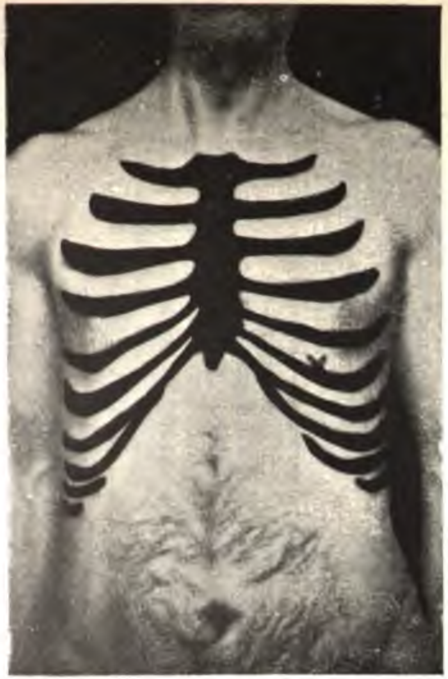
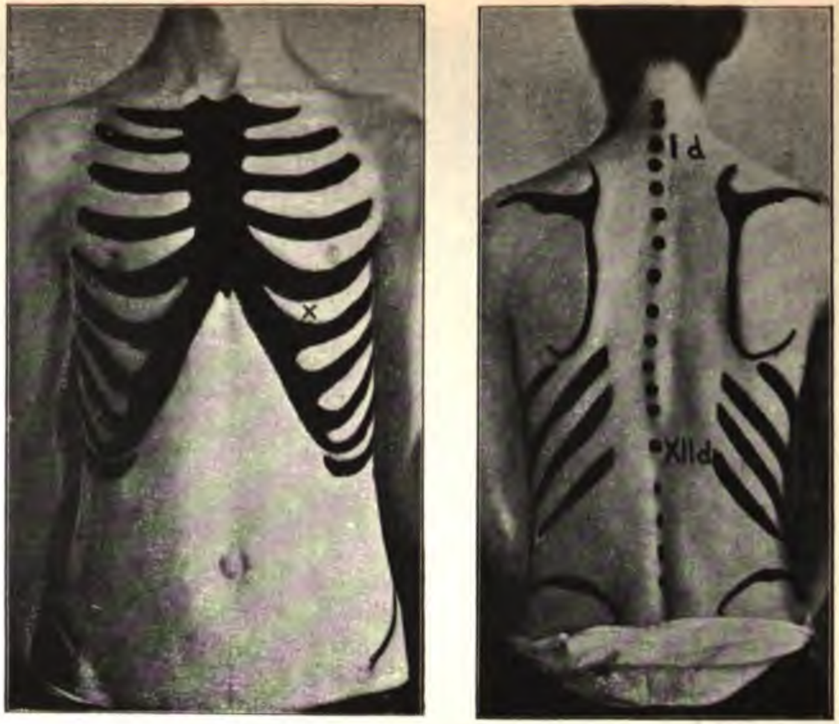


Fig. 16.—Emphysematous Chest
(not well marked)



Figs. 17 and 18.—Phthinoid Chest

The so-called phthinoid chest (figs. 17 and 18) corresponds more to the expiratory type; it is long and narrow, and is the chest of a person who is likely to be the subject of phthisis. The ribs are more nearly vertical. At the sides they may reach or even overlap the iliac crests; the lower intercostal spaces are diminished in width, and may be even actually obliterated by the approximation of the ribs. The subcostal angle is very acute.

Pigeon-breast—*pectus carinatum*—the result of frequent attacks of bronchitis in children, consists of an abrupt prominence of the rib cartilages and sternum.

Rickets may cause a great variety of changes of shape of the thorax; the commonest, however, is a general drawing in of the sides of the chest, and a consequent prominence of the anterior part. The lower parts are also prominent, owing to the usually distended state of the abdomen.

The cobbler's chest is one in which a deep depression exists at the lower part of the sternum.

Kyphosis, or exaggeration, of the thoracic curve may arise from a great variety of causes, amongst which may be mentioned caries of the spine, osteomalacia, spondylitis deformans, osteitis deformans, and the causes, whatever they may be, of lateral curvature.

Scoliosis, or lateral curvature, produces perhaps the greatest change of all in the shape of the chest; but here it is only possible to mention them, and to refer the reader to special books on surgery for the full description of their peculiarities; adding only this observation, that, great as the deformity is in these cases, it is not inconsistent with a perfectly healthy state of the thoracic viscera. The subject of scoliosis will, however, be again referred to when the deformity of the chest caused by the contractions due to pleurisy and empyema is dealt with.

Lastly, it must be pointed out that any swelling inside the thorax or abdomen, such as hypertrophied heart, pleural and pericardial effusions, tumours of the pleura, lungs, mediastinum, or liver, hydatids, ascites, aneurysms, &c., may cause local or general and more or less characteristic modifications of the shape of the thorax.

Intercostal vessels.—The *intercostal arteries* of the first two spaces come from the superior intercostal branch of the subclavian; the remainder come from the back of the aorta. They pass, with varying degrees of obliquity, to the lower borders of the ribs bounding the intercostal spaces above. Thus the upper ones begin by ascending for a considerable distance, while the lower ones are almost horizontal from the beginning. Near the spine they give off dorsal branches which pass backwards between the transverse processes. Near the angles of the ribs they reach the grooves on the inner surfaces of the ribs, the accompanying veins being above them and the nerves below. About the same point each artery gives off a collateral branch much smaller than the parent trunk, which runs along the upper border of the rib bounding the intercostal space below. Both vessels pass forwards to anastomose with the anterior intercostal branches of the internal mammary. On entering the space the arteries lie on the deep surface of the external intercostal muscles, and are covered by the pleura and the endothoracic fascia; afterwards they lie between the intercostal muscles, and at last penetrate the internal intercostals and lie between them and the pleura and endothoracic fascia. The seventh, eighth, ninth, and tenth anastomose with the musculo-phrenic branch of the internal mammary, the tenth and eleventh are continued into the abdominal wall. The intercostal arteries supply the muscles, the ribs and the subpleural tissue, and give off, besides those already mentioned, large lateral branches which pass outwards with the accompanying nerves about midway between the sternum and the spine.

The *intercostal veins* enter the azygos and the superior intercostal veins.

The *internal mammary* artery runs down behind the sterno-clavicular articulation, and the rib cartilages about half an inch from the sternum,[1] as far as the sixth intercostal space, where it divides into the superior epigastric, which enters the abdominal wall, and the musculo-phrenic, which passes outwards between the rib cartilages and the diaphragm, below the pleura, supplying the lower intercostal spaces, the abdominal wall, and the diaphragm. The internal mammary supplies two small intercostal branches to each space, perforating branches to the integuments, small sternal and mediastinal branches, and the comes nervi phrenici, which accompanies the phrenic nerve. Sometimes an unusual *lateral costal* branch, which may give rise to trouble in operating, leaves the internal mammary at its upper part, and passes downwards and outwards, crossing several of the ribs on their inner surface about midway between the spine and the sternum or somewhat farther forwards.

The *internal mammary veins* enter the innominate veins, the left passing into the left end of the left innominate, and the right

---

[1] Merkel, quoting from Sandmann (*Ueber das Verhältniss der Art. mammar. int. zum Brustbein*, Inauguraldiss., Königsberg, Nov. 16, 1894), gives the following table of distances of the internal mammary artery from the sternum:

1st intercostal space	11·1 mm.
2nd " "	15·3 "
3rd " "	15·6 "
4th " "	15·4 "
5th " "	16·9 "
6th " "	19·8 "

But I believe these figures are misleading, and are obtained by measuring along the intercostal spaces, which become more and more oblique below. My own observations show that, partly owing to the actual approach of the vessel to the middle line of the body, and partly owing to the greater width of the sternum below, the artery is separated by a shorter interval from the bone below than above.

This point, if true, is important, because it is in the fifth space that paracentesis pericardii is usually performed. I would warn the operator that unless he keeps his instrument quite close to the bone, he is very likely to injure the artery; and that, however careful he may be, he cannot be quite sure to avoid it.

either into the lower end of the right innominate or occasionally into the commencement of the superior cava.

The intercostal nerves.—The upper six run with the arteries near the lower borders of the ribs, and end by passing forwards close to the edge of the sternum as anterior cutaneous twigs. Each is united by two short branches with the ganglia of the sympathetic, which lie over the heads of the ribs, and each gives off a large lateral cutaneous branch.

The lower five intercostal nerves have at first relations similar to those of the upper six. They then pass through the origin of the diaphragm, on the deep surface of the costal cartilages, into the abdominal wall, where they supply both muscular and cutaneous branches. These and the twelfth dorsal (*subcostal*) nerve supply the greater part of the skin of the front and sides of the abdomen and a patch of skin on the outer part of the thigh. They are, therefore, worth the consideration of the physician when thinking about the referred pains associated with certain intrathoracic diseases, and of the surgeon when confronted with the question of dividing or saving some of these nerves in the course of an operation, or that of the removal or modification of a drainage tube which causes excessive pain.

Lymphatics of the thoracic wall.—Accompanying the intercostal bloodvessels in each space are two, or occasionally three, considerable lymphatic vessels, which collect tributaries from a plexus between the intercostal muscles. Passing backwards, these vessels enter the small *intercostal glands*, from one to three in number, at the hinder part of the space, the most constant being one close to the costovertebral articulation. The efferent vessels of the intercostal glands mostly pass transversely or ascend, to the thoracic duct; but the vessels from the lowest three or four spaces unite on each side in a trunk which descends on the bodies of the vertebræ to open into the receptaculum chyli.[1]

Other small lymphatic vessels, springing, in each space, from a plexus between the internal intercostal muscles and the pleura, ascend to a trunk, which runs forwards and inwards along the lower border of the rib and cartilage above the space to near the sternum, where it joins the longitudinal vessels accompanying the internal mammary artery and veins.[2]

Along the course of these vessels there are eight to ten *sternal glands*, which receive in addition some lymphatics from the fore part of the diaphragm, from the anterior abdominal wall, and, according to Sappey, from the liver also. Superiorly the longitudinal vessels terminate in the great lymphatic trunks at the root of the neck.

All the superficial lymphatics of the chest wall, including those of the muscles outside the ribs, and those of the mamma, are said to pass to the axillary lymphatic glands. No mention is made of communications between the lymphatics of the superficial muscles and those of the intercostal spaces. But the practical surgeon and the pathologist can scarcely doubt that such communications exist, in view of the frequency with which secondary subpleural nodules of cancer are met with on the same side of the body as that in which a scirrhus of the breast has invaded the pectoralis major.

---

[1] Sappey, *Traité*, p. 121, pl. xlv. fig. 6.

[2] *Ibid.* pl. xlv. fig. 5.

Mediastinum.—The already much curtailed intrathoracic space we have been describing is still further diminished by the irregular mass of viscera and other structures forming the mediastinum, which for the present purpose may be considered as a whole, without subdividing it into the various parts upon which it has been the habit of anatomists to exercise their ingenuity. For us it is the thick median partition extending from the spine to the front of the chest, and consisting of the heart and the pericardium, the great vessels, the trachea, the œsophagus, the thoracic duct, and numerous glands and nerves bound together by connective tissue, and covered on each side by the mediastinal pleura except at the place where the root of the lung is attached.

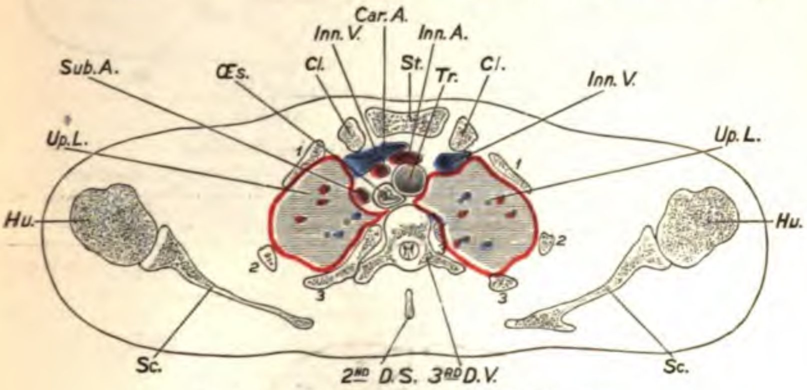


FIG. 19.—THROUGH THIRD DORSAL VERTEBRA

2nd *D S*, second dorsal spine ; 3rd *D V*, third dorsal vertebra ; *Sc*, scapula ; *Hu*, humerus ; *St*, sternum ; 1, 2, 3, ribs ; *Cl*, clavicle ; *Inn. V*, innominate veins ; *Inn. A*, innominate artery ; *Sub. A*, left subclavian artery ; *Car. A*, left common carotid artery ; *Tr*, trachea ; *Œs*, œsophagus ; *Up. L.* upper lobe of lung. The pleura is indicated by a red line.

(Figs. 19–23 are taken from Braune's well-known Atlas.)

This septum then is, roughly speaking, of a conical shape, the base being at the diaphragm on a level with the lower part of the eighth or the upper part of the ninth thoracic vertebra and the apex at the upper aperture of the thorax ; the base is attached to the diaphragm ; at the apex, which is slightly widened out laterally, the structures contained in it are continued upwards into the neck.

Behind, it is attached to the front of the spinal column, the attachment being deflected somewhat to the left at its lower part. This attachment is rather broader above, where it about equals the breadth of the vertebral bodies, than it is below. In front the mediastinum has a narrow linear attachment in, or slightly to the left of the middle line along the back of the sternum from the level of the second or third to the fifth costal cartilages; above this it widens out slightly behind the manubrium, and below it is also somewhat enlarged and deviates to the left of the median line.

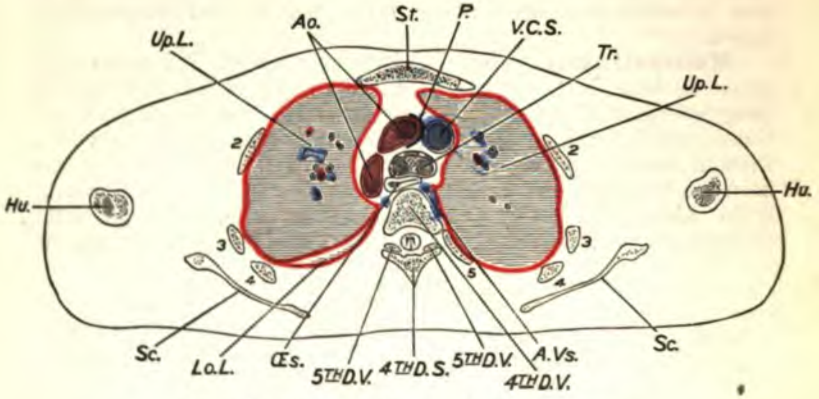


FIG. 20.—THROUGH FOURTH DORSAL VERTEBRA

4th *D V*, fourth dorsal vertebra; 4th *D S*, fourth dorsal spine; 5th *D V*, fifth dorsal articular processes; *St*, sternum; *Hu*, humerus; *Sc*, scapula; 2, 3, 4, 5, ribs; *Ao*, aorta; *V C S*, vena cava superior; *P*, pericardium; *Tr*, trachea; *Œs*, œsophagus; *Up. L*, upper lobes of lungs; *Lo. L*, lower lobe of left lung. The pleura is indicated by a red line.

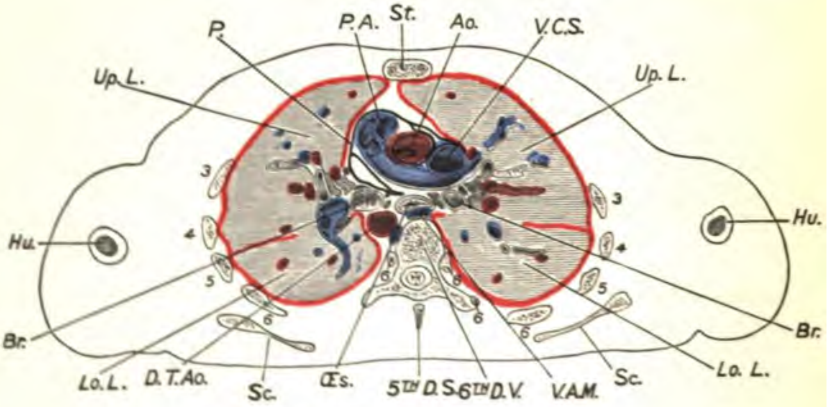


FIG. 21.—THROUGH SIXTH DORSAL VERTEBRA

6th *D V*, sixth dorsal vertebra; 5th *D S*, fifth dorsal spine; *Sc*, scapula; *Hu*, humerus; *St*, sternum; 3, 4, 5, 6, ribs; *P*, pericardium; *Ao*, aorta; *P A*, pulmonary artery; *V C S*, vena cava superior; *V A M*, vena azygos major; *Br*, main bronchi; *Œs*, œsophagus; *Up. L*, upper lobes of lungs; *Lo. L*, lower lobes of lungs. The pleura is indicated by a red line.

The presence of the heart in the lower half of the mediastinum gives to it the conical shape, and causes a very considerable projection towards the left side.

The shape of the mediastinum, and the difference in the manner in which it encroaches on the two pleural cavities, may be gathered from a study of frozen sections (figs. 19 to 23).

A glance at the attachments will explain the ease with which fluid or solid accumulations in either pleura may cause displacement of the mediastinum to one side or to the other; it will also indicate how a little shrinking or pushing away of the lung may cause the heart to come into contact with a much larger area of the chest wall than is natural, and perhaps give rise to the suspicion of a displacement that does not actually exist.

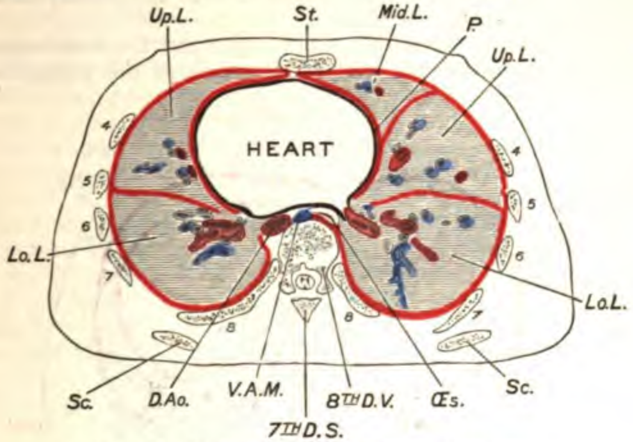


FIG. 22.—THROUGH EIGHTH DORSAL VERTEBRA

8th *D V*, eighth dorsal vertebra; 7th *D S*, seventh dorsal spine; *Sc*, scapula; *St*, sternum; 4, 5, 6, 7, 8, ribs; *D Ao*, descending thoracic aorta; *V A M*, vena azygos major; *Œs*, œsophagus; *P*, pericardium; *Up. L*, upper lobes of lungs; *Lo. L*, lower lobes of lungs; *Mid. L*, middle lobe of right lung. The pleura is indicated by a red line.

The pleura.—The shape of the cavity of the pleura corresponds to that of the contained lung, except on the left side in front of a portion of the pericardium, and on both sides below, where, for a variable distance, the diaphragm comes into contact with the chest wall. This unoccupied portion of the pleural cavity (*pleural sinus*), varying in extent with each act of respiration, is seen in figs. 28 to 31. It is only possible, by very careful auscultation and percussion, to estimate its extent. An exaggerated idea of it is obtained by post-mortem examinations, and it is indeed probable that the lung may reach to the bottom of it in extreme inspiration if the two surfaces have not, as is often the case, been more or less united by pleural adhesions.

The *visceral layer* of the pleura invests the lung completely, dipping to the bottom of the sulci between the lobes.

The *parietal layer* lines the ribs and the intercostal muscles, to which it is connected by a layer of areolar tissue, the so-called *endo-thoracic fascia*, which is the structure affected in the inflammatory condition known as *peripleuritis*. Near the spine the parietal pleura covers the intercostal vessels and nerves and the cord of the sympathetic, and is continued on to the sides of the bodies of the vertebræ, and the azygos veins to reach the sides of the mediastinum.

On the right side the *mediastinal pleura* is in relation above the root of the lung with the trachea and vagus nerve, the innominate artery and vein, the phrenic nerve, the superior vena cava with the arch of the azygos vein and the beginning of the arch of the aorta. Behind the root of the lung it is in contact with the œsophagus, and in front of it and below it with the pericardium and phrenic nerve (fig. 37).

On the left side it touches at the upper part the œsophagus, the left subclavian and carotid arteries, the arch of the aorta, the left phrenic and vagus nerves, and the beginning of the left innominate vein ; behind the root of the lung it is in contact with the descending thoracic aorta, and near the diaphragm with the œsophagus, and anteriorly with the pericardium and phrenic nerve (fig. 39).

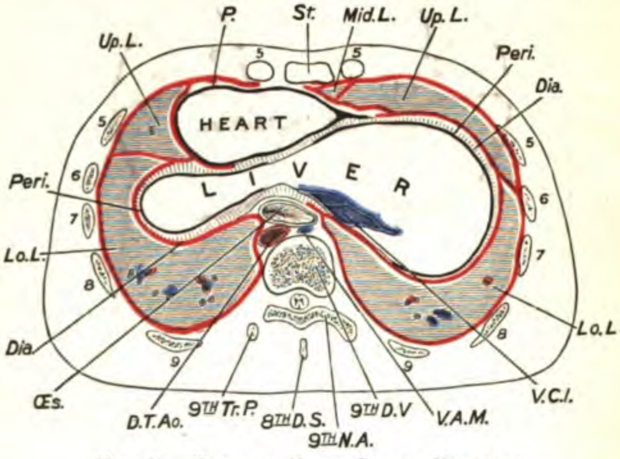


FIG. 23.—THROUGH NINTH DORSAL VERTEBRA

9th *D V*, ninth dorsal vertebra ; 9th *N A*, ninth neural arch ; 9th *Tr. P*, ninth transverse process ; 8th *D S*, eighth dorsal spine ; *St*, sternum ; 5, 6, 7, 8, 9, ribs and cartilages ; *D T Ao*, descending thoracic aorta ; *V C I*, vena cava inferior ; *V A M*, vena azygos major ; *Œs*, œsophagus ; *Dia*, diaphragm ; *P*, pericardium ; *Up. L*, upper lobes of lungs ; *Lo. L*, lower lobes of lungs ; *Mid. L*, middle lobe of right lung. The pleura is indicated by a red line.

The mediastinal and visceral layers of the pleura become continuous at the front and back of the roots of the lungs, and also by means of the *ligamentum latum pulmonis* (figs. 37 and 39) : a very short double layer of serous membrane, which occupies the interval between the root of the lung and the diaphragm, being attached externally to the inner surface of the lung, and on the inner side joining the mediastinum at the back of the pericardium. It is, however, not attached to the diaphragm.

The relation of the pleura to the œsophagus and the adjacent structures behind the roots of the lungs and the ligamenta lata pulmonum is worthy of attention. On the right side it dips in for a considerable distance between the back of the œsophagus and the front of the vertebræ and the vena azygos major, and sometimes extends for a short distance over the aorta below. On the left side there may be a smaller intrusion of the pleura between the œsophagus and the aorta. In this way the two pleuræ may come nearly into contact between the œsophagus and the aorta, about opposite the seventh, eighth, and ninth thoracic vertebræ.[1] This fact serves in part to explain not only the lateral mobility of the mediastinum, but possibly also the occasional extension of morbid processes from one pleura to the other.

Inferiorly, the parietal pleura is reflected on to the upper surface of the diaphragm, and above it extends through the upper aperture of the thorax into the neck.

This *cervical part of the pleura* is in contact on each side with the innominate vein, the scalenus anticus muscle, the subclavian artery, with its internal mammary and superior intercostal branches, and the lower trunk of the brachial plexus.

The pleuræ are not of precisely the same shape upon the two sides, the differences being principally accounted for by the projection of the heart to the left side. Roughly speaking, each consists of a hollow cone flattened, and, indeed, hollowed on the mesial aspect, with a rounded apex and a concave base, and extending much further downwards at the back than in front. The highest point of all is the same upon the two sides and corresponds to the neck of the first rib, and it does not reach above the level of this, the highest part of the first rib; but it will be remembered that the anterior end of the first rib is considerably below its head, and the pleura only projects very slightly above, or it might be said in front of the oblique plane in which the first rib lies.

The *apex of the pleura* is situated above the inner end of the clavicle, and reaches as a rule about three-quarters of an inch above this bone. The greater the obliquity of the first rib, the greater is the distance of the apex of the pleura above the clavicle, from which it follows that during full inspiration this distance is less than during expiration.

The pleura does not everywhere reach down as far as the attachment of the diaphragm, and therefore by no means corresponds to the lower margin of the bony thorax. The *lower limit of the pleura* is subject to considerable variation, but the following description may be taken to represent an average condition. On the right side, starting from the lower part of the sixth rib cartilage at its junction with the sternum or the sixth intercostal space, it passes obliquely to the junction of the seventh rib and its cartilage, then across the eighth, ninth, tenth, and eleventh ribs, at a gradually increasing distance from their distal ends, to reach the vertebral end of the twelfth rib (figs. 28 to 31). This line is, on the whole, convex downwards, but becomes almost horizontal behind the posterior fold of the axilla. In the middle of the axilla it is usually from two to three inches above the lower margin of the thorax. In this last

---

[1] Attention has been drawn to these recesses of the pleura by T. Jonnesco in Poirier's *Traité d'Anatomie Humaine*, tome iv. p. 185, 1895.

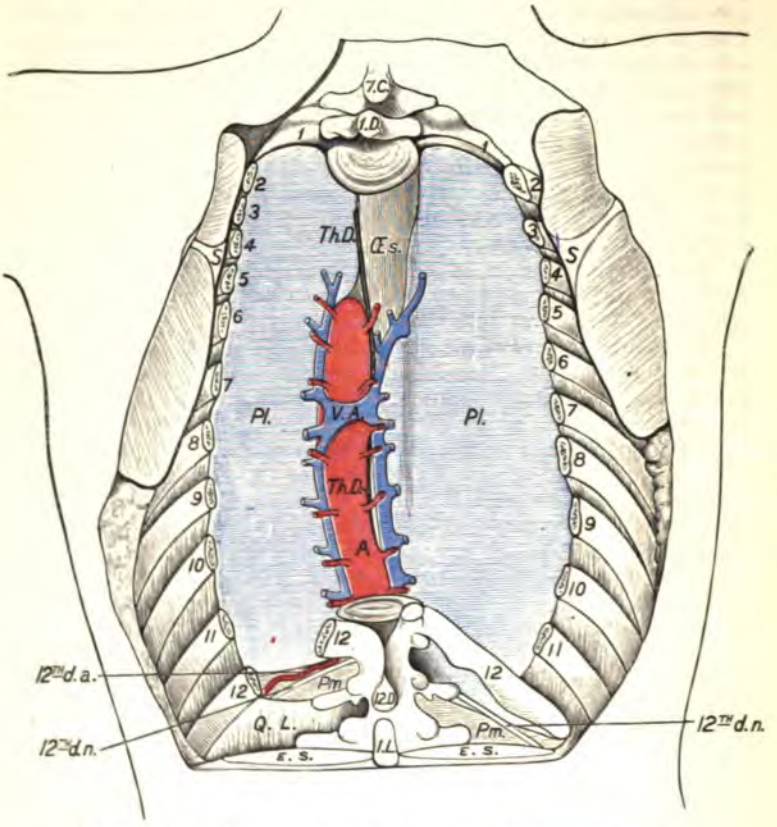


FIG. 24.—BACK VIEW OF CHEST OF AN OLD MAN, WITH SPINE AND RIBS REMOVED

The body was imbedded after hardening in formalin. The diaphragm was cut away on the right side. It will be observed that the twelfth rib was shorter on the right side than on the left. The pleura extended considerably below the twelfth rib on the right side. On the left side, though the twelfth rib was much longer, the pleura did not reach below its lower border. 1 to 12, ribs; *7 C*, seventh cervical vertebra; 1 *D*, first dorsal vertebra; 12 *D*, twelfth dorsal vertebra; 1 *L*, first lumbar vertebra; *S S*, scapulæ; *Œs*, œsophagus; *Th. D*, thoracic duct; *A*, aorta; *V A*, vena azygos; *Pl*, pleura; 12 *d a*, twelfth dorsal artery; 12 *d n*, twelfth dorsal nerve; *Pm*, peritoneum; *Q L*, quadratus lumborum; *E S*, erector spinæ

situation the variations are not great, and seldom exceed one finger's breadth. Near the spine, however, in some instances, especially when the twelfth rib is long and oblique, the pleura reaches down considerably lower, in extreme cases to the level of the first lumbar vertebra. It is, moreover, to be remarked that when the twelfth rib is very short, the pleura extends beyond the eleventh rib (which is then the last to be felt), and it projects in the same way beyond the thoracic margin. It thus appears that, while it is very unusual for it not to reach down as far as the upper border of the twelfth rib, unless it be obliterated by adhesions, it is most usual to find it extending for a greater or less distance below the thoracic margin, ıd it need not be pointed out how important this fact becomes in

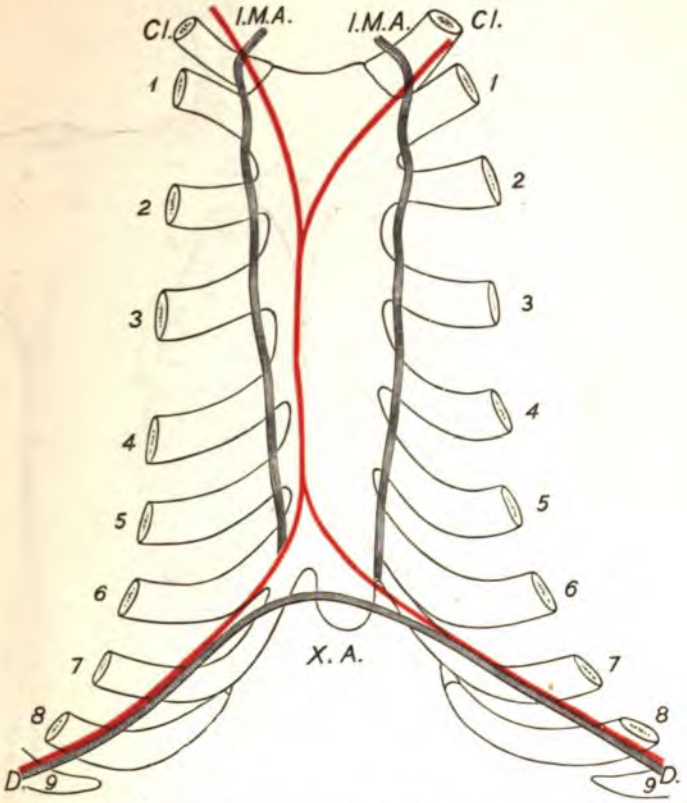


FIG. 25.—BACK VIEW OF STERNUM, SHOWING THE RELATIONS OF THE PLEURÆ TO THE FRONT OF THE CHEST. (The most usual condition)

1 to 9, ribs; *Cl*, clavicle; *X A*, xiphoid appendix; *D*, diaphragm; *I M A*, internal mammary artery. The pleura is marked by a red line.

to the junction of the seventh rib and its cartilage, eighth, ninth, tenth, and eleventh ribs, at a grad distance from their distal ends, to reach the verte twelfth rib (figs. 28 to 31). This line is, on th

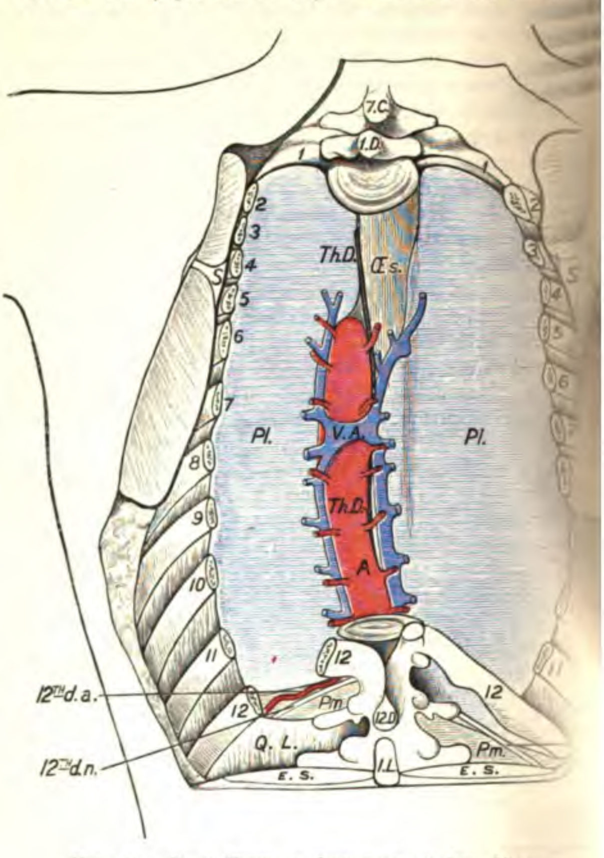


FIG. 24.—BACK VIEW OF CHEST OF AN OLD MAN,
RIBS REMOVED

The body was imbedded after hardening in formalin. The diaphrag right side. It will be observed that the twelfth rib was shorter on the left. The pleura extended considerably below the twelfth rib the left side, though the twelfth rib was much longer, the pleura lower border. 1 to 12, ribs; 7 *C*, seventh cervical vertebra; 1 *D*, 12 *D*, twelfth dorsal vertebra; 1 *L*, first lumbar vertebra; *S S*, scap *Th. D*, thoracic duct; *A*, aorta; *V A*, vena azygos; *Pl*, pleura; artery; 12 *d n*, twelfth dorsal nerve; *Pm*, peritoneum; *Q L*, quadr erector spinæ

downwards, but becomes almost horizontal behind of the axilla. In the middle of the axilla it is to three inches above the lower margin of the tho the left, so that while the behind the junction of the the left side ends close to pace (figs. 25, 26 and 28). pleura is greater than that

connection with operations requiring incisions up to, or the removal of, the last rib, or in wounds of the back (fig. 24).

The left pleura reaches about a finger's breadth lower than the right at the side, but differs little if at all from it posteriorly.

The line of the *anterior border of the pleura* starts from the apex, which is situated three-quarters of an inch above the clavicle between the two heads of the sterno-mastoid. At first slightly concave inwards, the line passes behind the sterno-clavicular articulation, from which point the anterior border is continued downwards not quite symmetrically on the two sides. At first they both slope inwards from the sterno-clavicular articulations to reach the middle line opposite the second costal cartilages, and the two pleuræ remain in contact for a short distance—that is, as far as the level of the fourth or fifth cartilages. They then diverge gradually outwards,

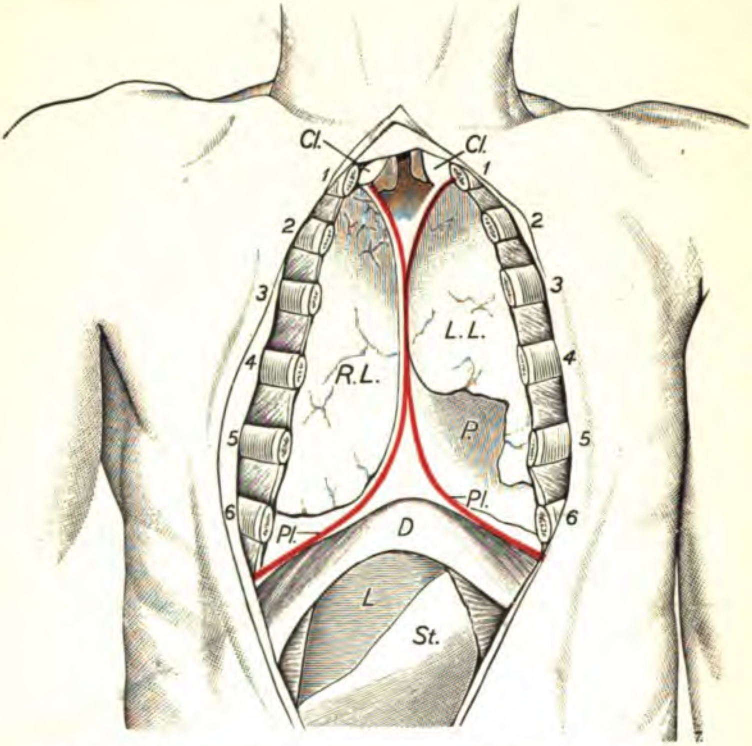


FIG. 26.—APPEARANCES SEEN IN THE CASE FROM WHICH THE STERNUM SHOWN IN FIG. 25 WAS REMOVED

1 to 6, ribs ; *Cl*, clavicles ; *D*, diaphragm ; *L*, liver ; *St*, stomach ; *R L*, right lung ; *L L*, left lung ; *Pl*, pleura ; *P*, pericardium.

the right rather more gradually than the left, so that while the anterior border of the right pleura ends behind the junction of the sixth cartilage and the sternum, that of the left side ends close to the sternum in the sixth intercostal space (figs. 25, 26 and 28). Occasionally the divergence of the left pleura is greater than that here described, the serous membrane following more exactly the line of the lung (fig. 27). This divergence to the left depends upon the presence of the heart, and it is of importance because it sometimes leaves a small part of the pericardium at the extreme inner end of the fifth intercostal space uncovered by pleura—the only situation in which it is possible to puncture the pericardium without previously

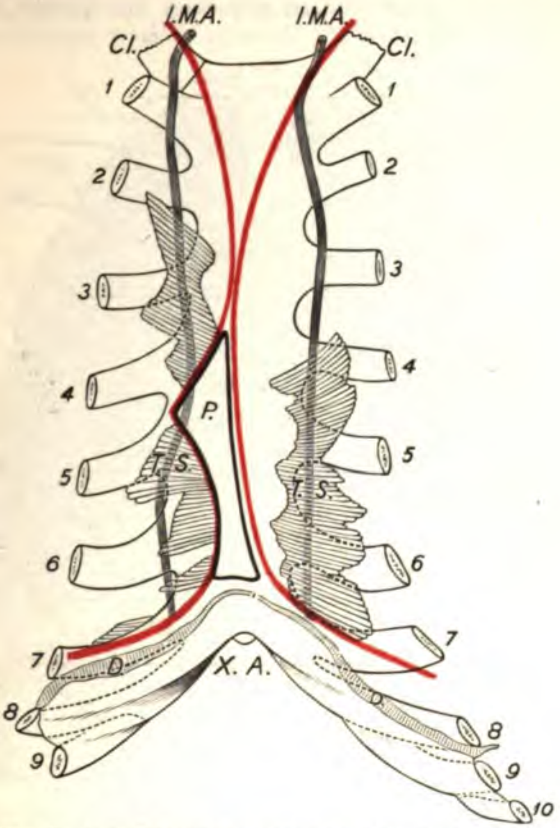


Fig. 27.—Back of Sternum, showing the less usual arrangement of the Anterior Border of the Left Pleura

1 to 10, ribs; *X A*, xiphoid appendix; *Cl*, clavicle; *T S*, triangularis sterni; *I M A*, Internal mammary artery; *D*, diaphragm; *P*, pericardium. The pleura is marked by a red line.

wounding the pleura; but it must not be forgotten that even this small area of pericardium uncovered by pleura is not by any means always present. There is, however, considerable variation in the arrangement of the anterior borders of the pleuræ, a common modification consisting in the encroachment of the right over the left. The right pleura may reach the left border of the sternum, or even further. On the other hand, in rare cases the opposite condition may exist, and the right pleura may not even extend as far as the right border of the sternum. On the whole, it will be observed that the right pleura is shorter but broader than the left.

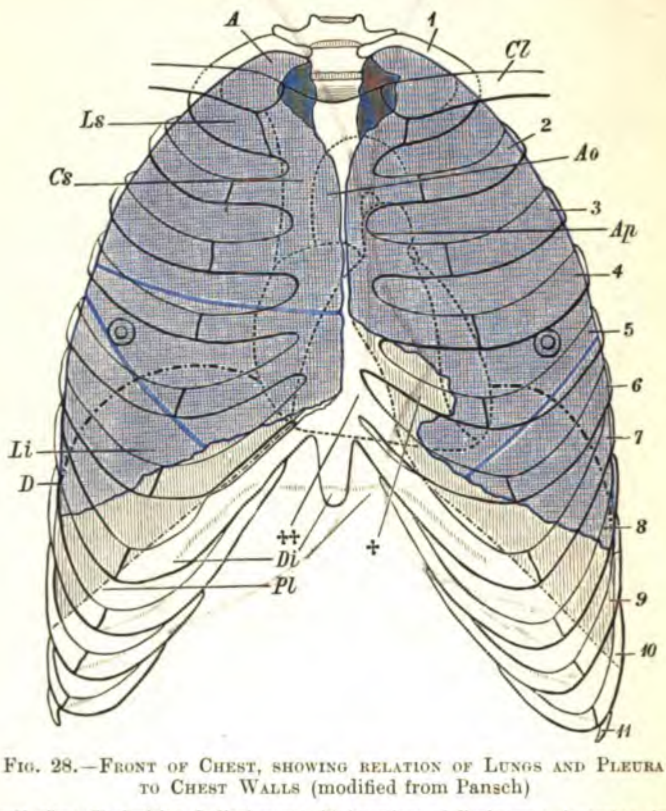


FIG. 28.—FRONT OF CHEST, SHOWING RELATION OF LUNGS AND PLEURA TO CHEST WALLS (modified from Pansch)

1 to 11, ribs; *Cl*, clavicle; *D*, diaphragm; *Di*, insertions of diaphragm; *Ao*, aorta; *Ap*, pulmonary artery; ✠, incisura cardiaca; ✠ ✠, pericardium in contact with chest wall; *Ls*, upper lobe of lung; *Lm*, middle lobe of right lung; *Li*, lower lobe of lung.

The lungs.—The lower margins of the lungs do not reach so far down as the bottom of the pleural cavity, unless it be in forcible inspiration, and the left lung extends a finger's breadth lower than the right. Starting on the right side from the junction of the sixth cartilage with the sternum, this margin passes behind the junction of the sixth rib and its cartilage, and, pursuing a course at first oblique and then almost horizontal, reaches the vertebral end of the eleventh rib (fig. 29).

The apex and anterior border of the right lung fit exactly into the corresponding parts of the pleura. The left lung, after completely filling the pleura as far as the level of the fourth cartilage, diverges from it in front of the heart to form what is known as the V-shaped notch (*incisura cardiaca*, figs. 26 and 28). The anterior border must therefore be marked on the surface as leaving the middle line opposite the fourth cartilage, and passing obliquely downwards, almost to the apex of the heart; from this

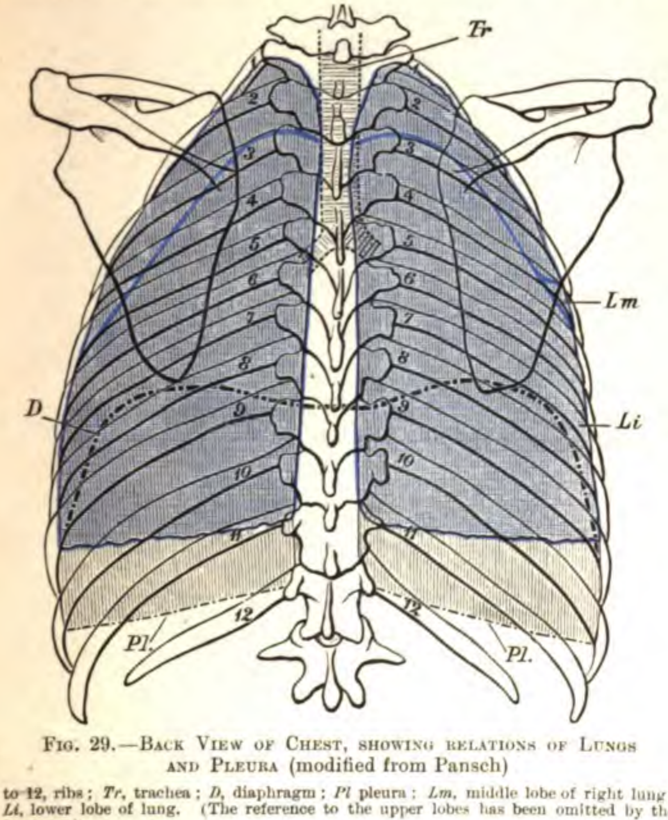


FIG. 29.—BACK VIEW OF CHEST, SHOWING RELATIONS OF LUNGS AND PLEURA (modified from Pansch)

1 to 12, ribs; *Tr*, trachea; *D*, diaphragm; *Pl* pleura; *Lm*, middle lobe of right lung; *Li*, lower lobe of lung. (The reference to the upper lobes has been omitted by the engraver.)

oint it returns towards the sternum, and joins the lower border at a le distance from the sternum, either behind the sixth cartilage th intercostal space. Posteriorly, the lungs are separated corresponding to the width of the vertebral bodies.

The great fissure which separates the lower lobe from the rest of the lung follows a line which runs from the second dorsal spine (fig. 29), at first horizontally outwards and then obliquely downwards to join the lower border of the lung opposite the middle of the sixth cartilage or fifth intercostal space just above this point, and usually passes just below the nipple (fig. 28). The smaller fissure which separates the upper and the middle lobes of the right lung leaves this line about the middle and joins the anterior border opposite the fourth cartilage (figs. 29, 30 and 28). For the greater part of its extent it follows approximately the line of the fourth rib and cartilage (fig. 36).

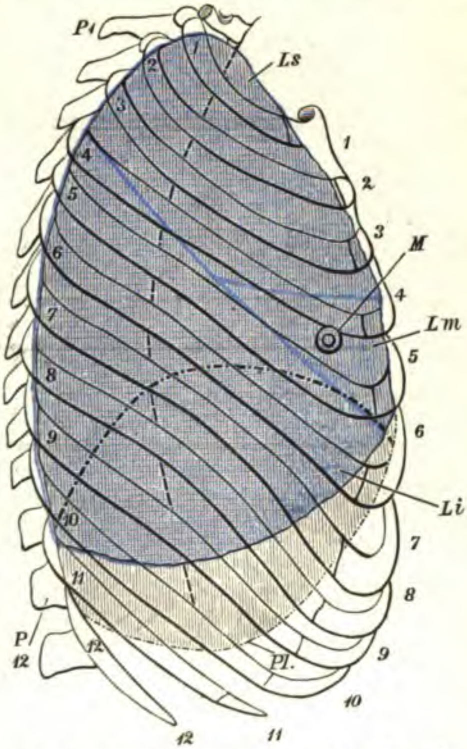


FIG. 30.—RIGHT SIDE OF CHEST (after Pansch)

P 1, spinous process of first thoracic vertebra; P 12, spinous process of twelfth thoracic vertebra; 1 to 12, ribs; M, nipple; Ls, upper lobe of lung; Lm, middle lobe of lung; Li, lower lobe of lung; Pl, Pleura.

Very little idea of the *shape of the healthy lung* is obtained from its appearance in the post-mortem room. A better notion is given by one that is solidified by disease, and a still better one from casts obtained by hardening the whole body and pouring plaster of Paris into the cavities which are left by removing one organ after another. But whatever method of study is followed, it is impossible to obtain an actual view of the lung in anything except a state of expiration, unless it be artificially inflated, and as the degree of inflation which is permissible must be left to the judgment of the observer, no very definite results can be anticipated.

It must be imagined, then, as filling the greater part of the pleural cavity and as taking its shape from the surrounding parts. Thus the rounded costal surface receives impressions from the ribs, and in front of the apex there is a groove corresponding to the subclavian vessels. The inner surface is moulded on to all the irregularities of the bodies of the vertebræ and the structures contained in the mediastinum, and the lower surface fits on to the smooth surface of the diaphragm, as will be explained in detail on pages 33 to 35.

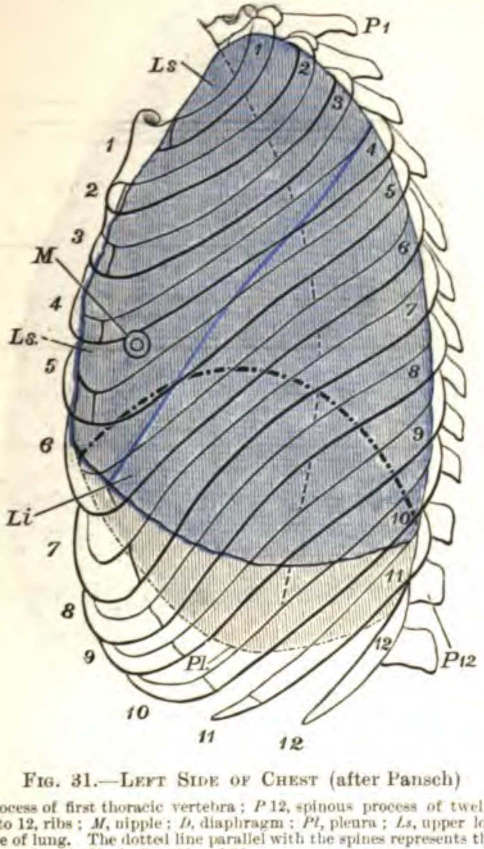


FIG. 31.—LEFT SIDE OF CHEST (after Pansch)

P 1, spinous process of first thoracic vertebra; *P* 12, spinous process of twelfth thoracic vertebra; 1 to 12, ribs; *M*, nipple; *D*, diaphragm; *Pl*, pleura; *Ls*, upper lobe of lung; *Li*, lower lobe of lung. The dotted line parallel with the spines represents the line of the front of the vertebral bodies in figs. 30 and 31.

Little is gained by ascribing an exact geometrical figure to the shape of the lung. It is enough to recognise for descriptive purposes an apex and a base, an inner and an outer surface, and an anterior and a posterior border. By the latter is meant the rounded part that fits into the hollow formed by the angles of the ribs and which serves in a very indefinite manner to separate the inner from the outer surfaces. The anterior border is, on the other hand, very thin, except at the upper part. Beginning at the apex it is rounded and not very definite and is interrupted by the groove for the subclavian vessels. It takes a curved course round the structures passing from the thorax into the neck with the concavity inwards, and then it becomes very thin opposite the sterno-clavicular articulation, and runs down in the line described on p. 29 to join the sharp margin which separates the base from the outer surface of the lung. This lower border is thin for the most part, it sweeps round the lower surface of the lung with an even curve until it reaches the bodies of the vertebræ, after which it becomes irregular

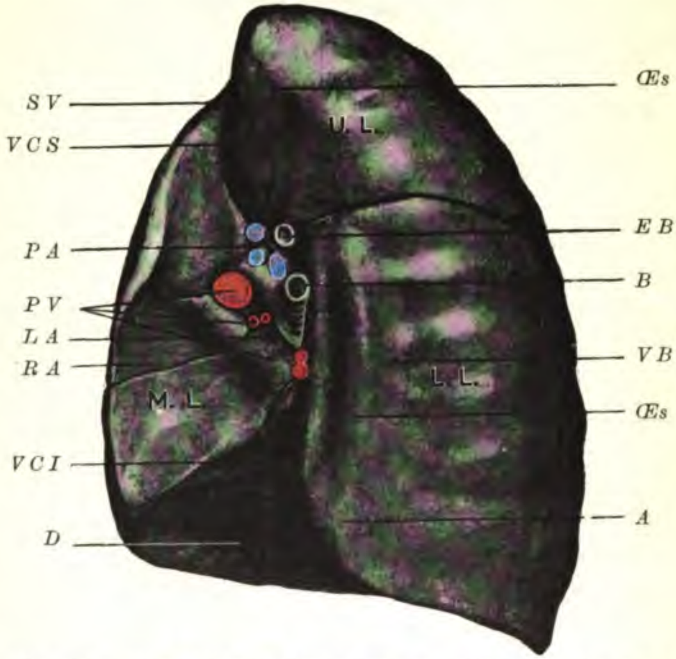


Fig. 32.—Inner Aspect of Right Lung (from His's Model), showing the parts in contact with the Lung

U L, upper lobe of lung; *L L*, lower lobe of lung; *M L*, middle lobe of lung; *S V*, subclavian vessels; *V C S*, vena cava superior; *Œs*, œsophagus; *L A*, left auricle; *R A*, right auricle; *V B*, vertebral bodies; *A*, aorta; *B*, main bronchus; *E B*, eparterial bronchus; *P A*, pulmonary artery; *P V*, pulmonary veins; *D*, diaphragm; *V C I*, vena cava inferior.

in shape as it is moulded round the vertebral bodies, the aorta and œsophagus, and on the right side round the right auricle, as the vena cava inferior enters it; and on the left side round the left ventricle and the apex of the heart.

On looking at the *mesial aspect of the lung* (figs. 32 and 33), it is seen that there is a considerable difference between the inner surfaces of the two organs. Each is roughly triangular in shape, and is divided into two unequal parts by a curved line almost parallel with the posterior border. The part behind this line is much larger than that in front of it, and is marked by impressions corresponding to the bodies of the vertebræ and the ribs. The smaller anterior portion is deeply hollowed, and much more deeply on the left side than on the right owing to the presence of the heart. At the upper and posterior part of this depression is the irregularly triangular or oval-shaped hilum at which the bronchus and vessels pass into and from the lung. Running upwards from the upper and anterior part of the cardiac impression on the right side is a deep groove which

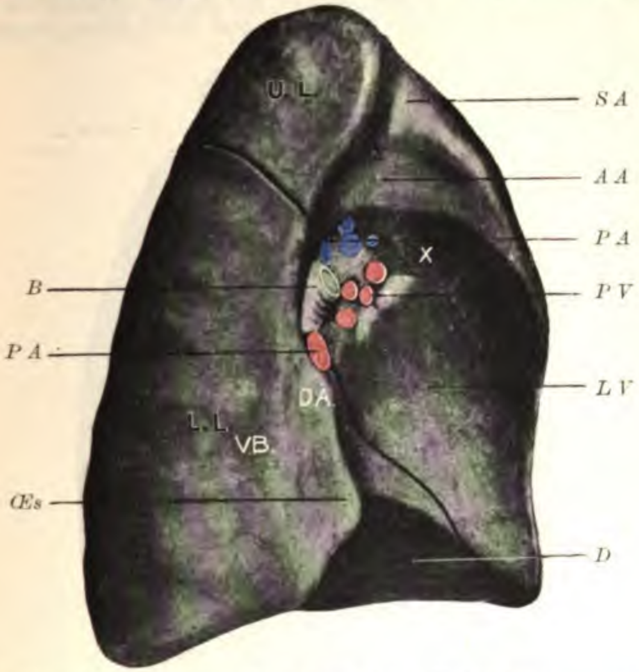


FIG. 33.—INNER ASPECT OF LEFT LUNG (FROM HIS'S MODEL), SHOWING MARKS OF STRUCTURES IN CONTACT WITH LUNG

U L, upper lobe of lung; *L L*, lower lobe of lung; *S V*, subclavian vessels; *A A*, arch of aorta; *X*, conus arteriosus and left auricular appendix; *D A*, descending aorta; *Œs*, œsophagus; *D*, diaphragm; *L V*, left ventricle; *B*, bronchus; *P A*, pulmonary artery; *P V*, pulmonary vein; *V B*, bodies of vertebræ.

obvious in connection with the appreciation of the progress of pneumonia, phthisis, and other diseases, and the diagnosis of solid affections of the lower lobe from accumulations of fluid in the pleura.

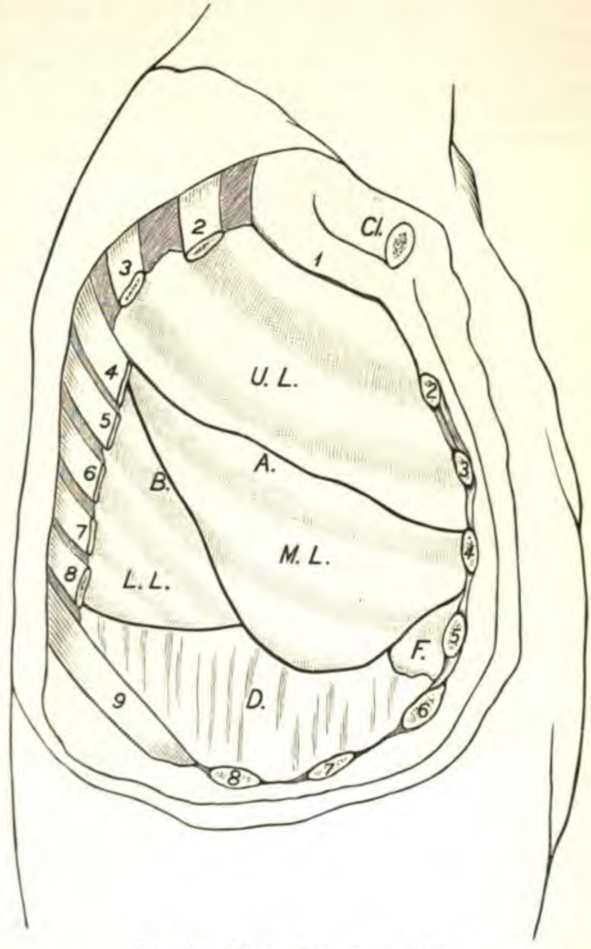


FIG. 36.—OUTER ASPECT OF CHEST

The body was injected with formalin and the ribs removed. 1 to 9, ribs; *Cl*, clavicle; *F*, pellet of fat; *D*, diaphragm; *U L*, upper lobe of lung; *M L*, middle lobe of lung; *L L*, lower lobe of lung; *A*, fissure between upper and middle lobes; *B*, fissure between middle and lower lobes.

A transverse section of the *root of the lung* is of an irregular oval shape becoming narrower below (figs. 37 and 39, 32 and 33). The various structures entering and leaving the lung are firmly bound together by dense areolar tissue, containing rather large lymphatic glands, which are wedged in amongst the bronchi and vessels, and assume very irregular shapes in consequence. On the left side, the bronchus enters about the middle of the posterior part of the hilum, and one vein is in front of it. The other vein is directly

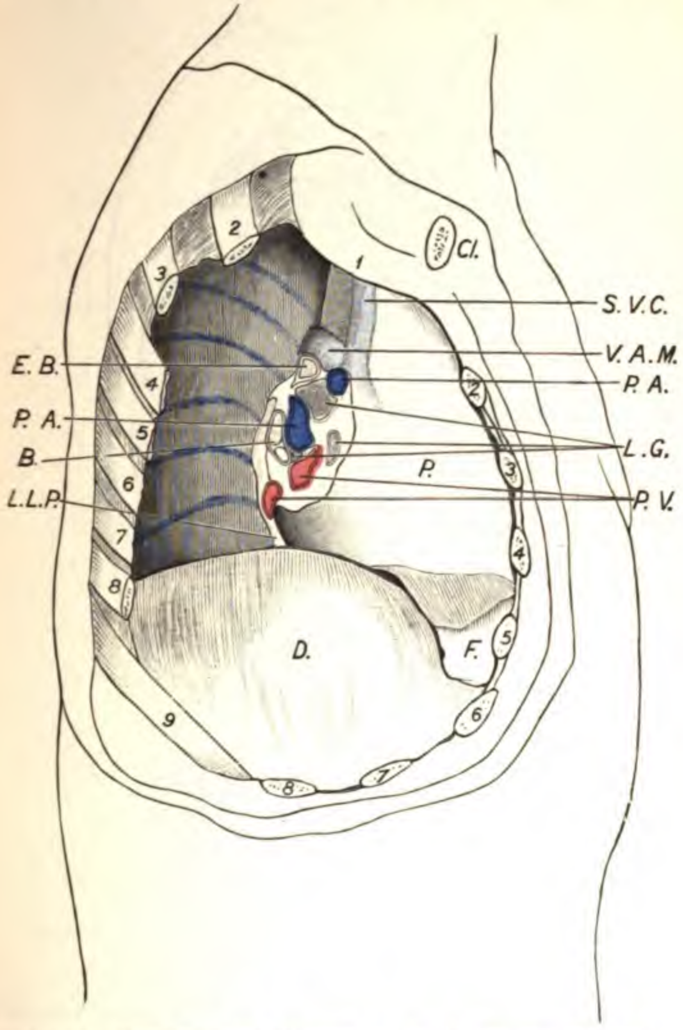


Fig. 37.—From the same Subject as Fig. 36, showing the Root of the Lung and the Mediastinum

1 to 9, ribs; *Cl*, clavicle; *D*, diaphragm; *S V C*, superior vena cava; *V A M*, vena azygos major; *E B*, eparterial bronchus; *L G*, lymphatic glands; *P A*, pulmonary artery; *B*, main bronchus; *P V*, pulmonary veins; *L L P*, ligamentum latum pulmonis; *P*, pericardium; *F*, lappet of fat.

below it. The artery is the highest structure in the hilum. On the right side the arrangement is modified by the presence of the eparterial bronchus (page 43, figs. 40 to 42), which, with its accompanying artery, enters the lung above the main branch of the pulmonary artery.

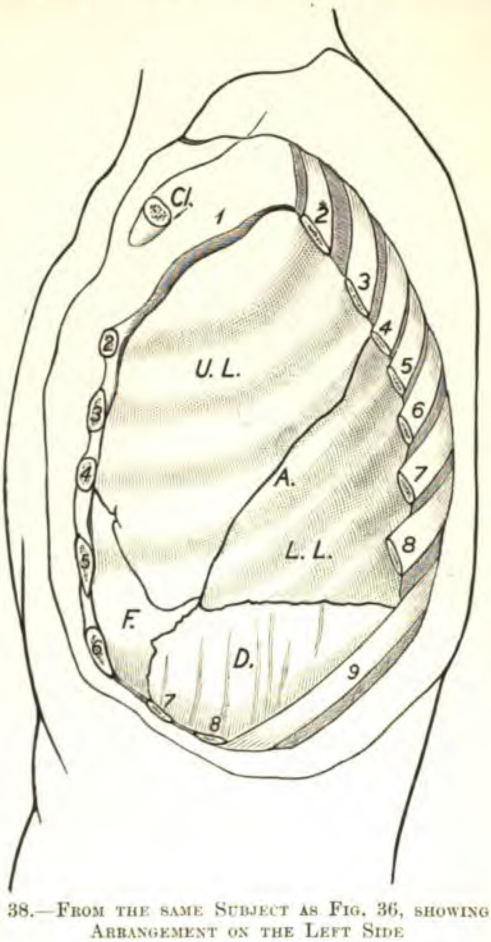


FIG. 38.—FROM THE SAME SUBJECT AS FIG. 36, SHOWING THE ARRANGEMENT ON THE LEFT SIDE

1 to 9, ribs; *Cl*, clavicle; *F*, lappet of fat; *D*, diaphragm; *A*, fissure between lobes; *U L*, upper lobe of lung; *L L*, lower lobe of lung.

Immediately after entering the lung, the bronchus and the vessels subdivide. A little dissection then shows the following arrangement. The branches of vein occupy an oblique line running downwards and backwards at the lower part, the bronchi a parallel

line above them, and the branches of artery are above the bronchi; but the eparterial bronchus still maintains the highest position.

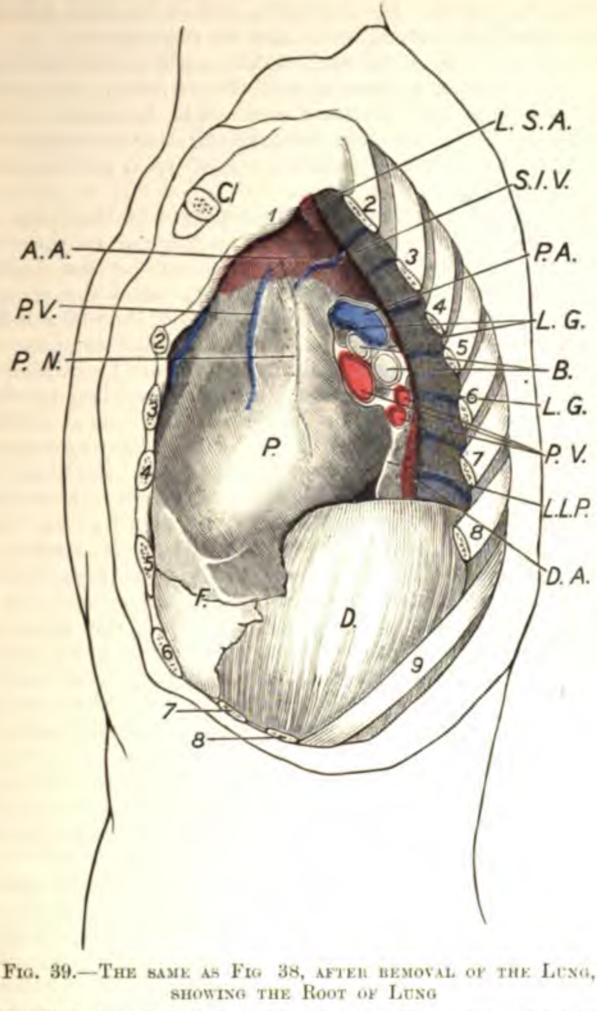


Fig. 39.—The same as Fig 38, after removal of the Lung, showing the Root of Lung

1 to 9 ribs; *Cl*, clavicle; *D*, diaphragm; *F*, lappet of fat; *P*, pericardium; *L S A*, left subclavian artery; *A A*, arch of aorta; *D A*, descending aorta; *S I V*, superior intercostal vein; *P V*, pericardial vein; *P N*, phrenic nerve; *P A*, pulmonary artery; *P V*, pulmonary veins; *B*, bronchus dividing; *L G*, lymphatic glands; *L L P*, ligamentum latum pulmonis.

The roots of the lungs are opposite the fifth, sixth, seventh, and sometimes the eighth thoracic vertebræ.

In front of the right is the superior vena cava and part of the right auricle and the phrenic nerve. The vena azygos major arches above it. Behind it is the œsophagus and the vagus nerve.

In front of the root of the left lung is part of the left auricle and the phrenic nerve. Above it is the arch of the aorta, and behind it is the descending thoracic aorta and the vagus nerve.

The close relation of the arch of the aorta to the left bronchus will be remembered in connection with the not infrequent occurrence of partial or complete obstruction of this bronchus by aortic aneurysms, and the consequent development of acute bronchiectasis.

The relation to it of the œsophagus is of equal pathological importance.

Movements of the lung.—The movements of the lung during respiration are of two sorts, a general expansion as the walls of the chest expand, and a downward movement as the diaphragm contracts. During the process of the expansion of the chest walls the ribs are raised. From both of these causes, therefore, it follows that the surface of the lung glides downwards over the inner surface of the chest walls during inspiration, an obvious fact which apparently has only comparatively recently been noticed by physiologists, although the presence of friction fremitus in the course of pleurisy gives a constant object lesson of its truth. The movements of different parts of the lungs are subject to great variations. Thus the expansion of the upper part is more marked in women than in men, and in the former is probably increased by the habit of restraining the movements of the lower ribs by tight corsets. It is in tight-laced women that the heaving of the upper part of the chest during inspiration is most obvious. In men, on the other hand, quiet respiration is mostly abdominal. A rigid chest restrains the movements of the upper parts of the lungs, and it is probable that the very shape of the phthinoid chest may have something to do with the predilection shown by tubercle to affect the apex of the lung. The root of the lung is clearly by far the most fixed part of the organ.

The trachea.—Starting in the middle line opposite the lower border of the sixth cervical vertebra, the trachea passes downwards with a very slight inclination to the right at the lower part, and ends at the level of the disc between the fourth and the fifth thoracic vertebræ. It also follows the direction of the thoracic spine from which it is separated by the flattened œsophagus, and thus reaches further and further from the anterior surface of the body as it descends. Thus, whereas at the commencement it is almost subcutaneous, it is separated from the skin by a distance of one and three-quarter inch or more at the level of the top of the sternum in a well-grown man (fig. 4), and it is usually from two and a half to three inches from the surface at its bifurcation. The bifurcation of the trachea is just above the level of the junction between the manubrium sterni and the gladiolus, and corresponds behind with that of the spines of the scapulæ (fig. 29).

When the face looks directly forward the cricoid cartilage is from two to three inches above the top of the sternum; if the head be thrown back the distance between the two is increased by an inch, and when the chin is lowered the cricoid cartilage may be made to approximate or even pass behind the top of the sternum. Thus in cases of ankylosed and flexed cervical spine, as for example after caries or some rheumatic affections, there may be absolutely no trachea in the neck available for tracheotomy. In the infant the cervical part of the trachea is relatively longer than in adult life, and the bifurcation of the trachea is one vertebra higher at birth than during adult life (Symington).

The *length of the trachea* does not bear any constant relation to the height of the body. It is difficult to measure it accurately unless the measurement be taken to the angle between the divergent bronchi, which seems to give rather more to the trachea than actually belongs to it. It is said to be on an average about four inches to four and a half inches (10 to 11 cm.) long; but the variations are so great that these numbers are not of any great value, and, moreover, the length varies to some extent with the position of the neck—Braune says as much as an inch. It does not become obviously curved, even when the neck is greatly flexed or extended.[1]

Its *shape in section* is roughly that of a circle with the posterior part cut off, the shape being maintained by the sixteen or twenty cartilaginous rings embedded in its wall. Metal casts of the tube are nearly round because the weight of the metal bulges the posterior wall backwards, but in frozen sections the posterior wall bulges slightly into the trachea, though presumably during life it is nearly flat.

Its outside measurement is said to be from three-quarters of an inch to one inch.[2] It has been stated (Aeby) that the trachea gradually increases in calibre from above downwards, and by others (Braune and Stahl) that it is smallest at its commencement, but gradually increases in size to within about three centimetres of the bifurcation, where it again enlarges. At the lower end it becomes flattened from before back.

In the last edition of Quain's 'Anatomy' it is stated by J. Symington that in an infant six months old the trachea will admit a tube 4 mm. in diameter, at two years one of 5 mm., and at six years of age one of 6 or 7 mm.

In the female the diameter of the trachea is smaller than in the male, though there is not much, if any, difference in the average length. The cartilages do not as a rule begin to ossify before the age of sixty, while in man they often show this change at forty years of age.

The trachea rests on the œsophagus and the vertebral column.

---

[1] *Topograph. Anat. Atlas*, s. 21; Leipsic, 1875.

[2] Lejars, *Revue de Chirurgie*, 10 avril, 1891, onzième année, No. IV. p. 336: sag. diam. 11 mm.; tr. diameter 12·5 mm. Marc Sée, *Gazette Hebdom. de Méd. et de Chirurg.* 2, mai, 1884, tome xxi, No. 18, p. 294: average diameter, men, 18 mm.; women, 14·5 mm. Aeby: sag. diam. 17·6 mm.; tr. diameter, 16·6 These measurements apply to the lumen of the tube, and are not e measurements.

At the beginning the œsophagus is directly behind it, but at the lower part of the neck the œsophagus inclines so much to the left that the back of the trachea is usually applied directly to the front of the vertebral column.

In the neck it has the sternohyoid and sternothyroid muscles, the thyroid gland and the inferior thyroid veins, and possibly the thyroidea ima artery in front of it, while to each side are the carotid arteries and the recurrent laryngeal nerves, the latter being placed laterally between the trachea and the œsophagus.

In the thorax it lies behind the manubrium sterni, and is crossed by the arch of the aorta and by the left innominate vein. The innominate artery and the left carotid are at first in front and then somewhat to the sides of the trachea. The pleura and the vagus touch it on the right side. On the left side it is in contact with the left subclavian artery, the arch of the aorta, and some of the cardiac nerves. The deep cardiac plexus of nerves is situated between the arch of the aorta and the bifurcation of the trachea.

The bronchi and pulmonary arteries.—There is a wide difference between the modern way of describing the course and method of subdivision of the bronchial tubes from that which was adopted some time ago. This is principally due to the observations of Aeby, in great part confirmed by later anatomists. It partly also depends on the fact that there is really a considerable variety in the length and relative position of the main bronchi, as may be seen by studying a number of metal casts. It used to be said that the right bronchus came off more abruptly from the trachea than the left, a mistake which arose from the fact that its first branch comes off earlier than the first branch on the other side, and thus apparently alters the direction of the tube. It is now recognised that the right bronchus usually follows more nearly than the left the course of the trachea, which it will be remembered is directed slightly to the right at the lower end (figs. 29, 40 and 41). In many cases the trachea and the right bronchus are almost absolutely in the same line. The angle between the diverging bronchi is sometimes as wide as a right angle, but more frequently is decidedly acute (figs. 40 and 41).

It also used to be said that the bronchi divided and subdivided dichotomously, and there was a considerable excuse for this description, because the branches given off are often so nearly of the same size as the main stem that the appearance of dichotomous division is closely simulated. The study of comparative anatomy and of metallic casts has convinced almost everyone, with certain exceptions, such as that of Dr. Ewart, that, as in other animals, there is really one main stem starting from the bifurcation of the trachea and running down the lung to end in the thin posterior part between the diaphragm and the ribs, about three inches from the middle line of the body. This main stem is thus directed downwards and backwards, on the left side it is also curved considerably with the convexity outwards, on account of the presence of the heart.

From these main stems are given off lateral branches, the arrangement of which is not precisely the same on the two sides. Before describing them it will be necessary to say a word about the relation of the pulmonary artery and its subdivisions to the bronchi.

The main bronchial stem enters the hilum of the lung at the upper and posterior part. The *main branch of the pulmonary artery* enters at the upper part and in front, and, crossing the bronchus, reaches its posterior aspect. Throughout the lung the branches of the pulmonary artery accompany the bronchi, the smaller arteries lying behind the smaller bronchi. An account of the arrangement of the pulmonary veins in the lung is given after the completion of the description of that of the bronchi.

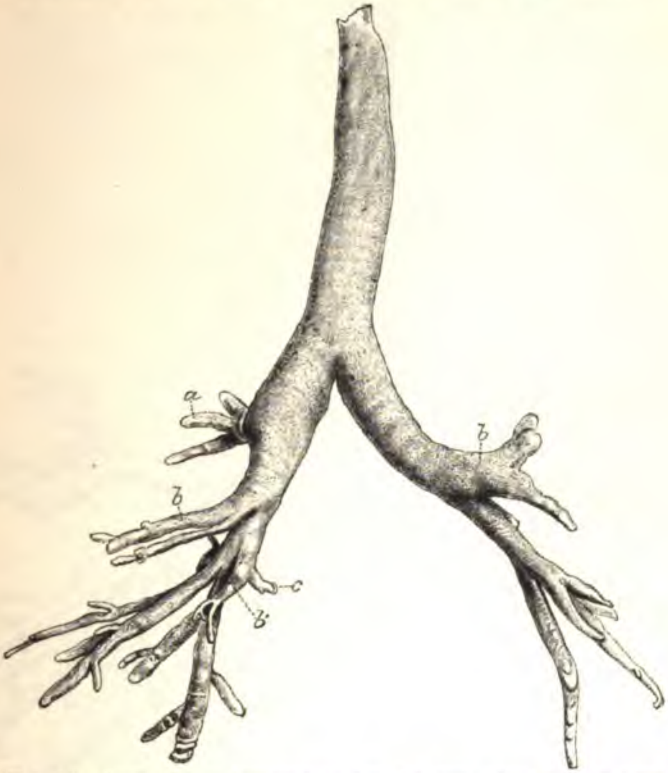


FIG. 40.—CAST OF THE INTERIOR OF THE TRACHEA AND BRONCHI, WITH THEIR CHIEF RAMIFICATIONS WITHIN THE LUNG. (Aeby)

This cast shows a type of division frequently met with, the right bronchus being almost in continuation of the line of the trachea. *a*, eparterial branch ; *b*, *c*, hyparterial branches (ventral and dorsal).

The main bronchus on the right side gives off a large branch to the upper lobe of the lung, before it is crossed by the pulmonary artery, which is thus called the *eparterial bronchus*. On the left side there is no such arrangement. It follows from this that the right main undivided stem is shorter than the left, and on the left side all the branches are hyparterial.

The lateral branches are more or less symmetrical on the two sides and are arranged in two sets, ventral and dorsal—four of each—of which the ventral or outer are much the larger, and, indeed, some of them are as large as the continuation of the main trunk.

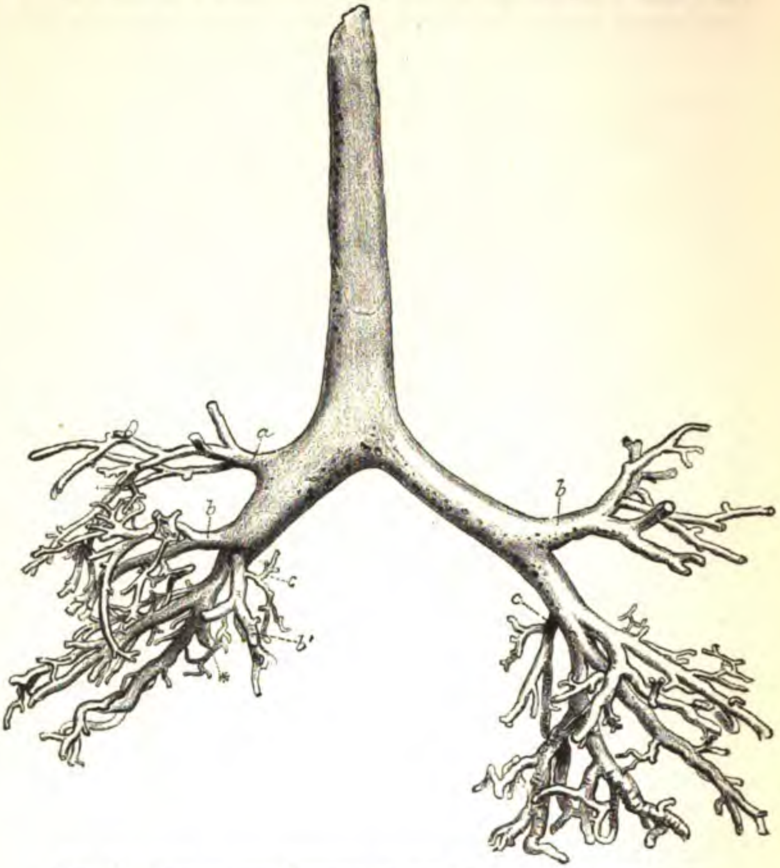


FIG. 41.—CAST OF THE INTERIOR OF THE TRACHEA AND BRONCHI, WITH THEIR CHIEF RAMIFICATIONS WITHIN THE LUNGS. (Aeby)

This cast shows a type of division less frequent than the last, the right and left bronchi being at about a right angle with one another. *a*. eparterial branch ; *b*, ventral hyparterial branches ; *b'*, accessory (azygos) branch ; *c*, dorsal hyparterial branches.

Besides the dorsal and ventral, there is a somewhat irregular series of branches called by Aeby accessory bronchi. They usually arise from the front, being intermediate in position between the dorsal and ventral branches. One of these near the second left ventral branch on the right side was called the *heart bronchus* by Aeby, and it is interesting because in some animals it supplies a small separate lobe situated behind the pericardium which has received the name of the *azygos lobe*.

Hasse points out that the larger branches of the bronchi in the upper and middle lobes of the right lung and the upper lobe of the left lung are directed outwards, upwards, and forwards, while those of the lower lobe of both lungs are directed downwards, inwards, and backwards, and says that these directions agree with the movements of the chest walls, which in the upper and anterior part expand in an upward, forward, and outward direction, while owing

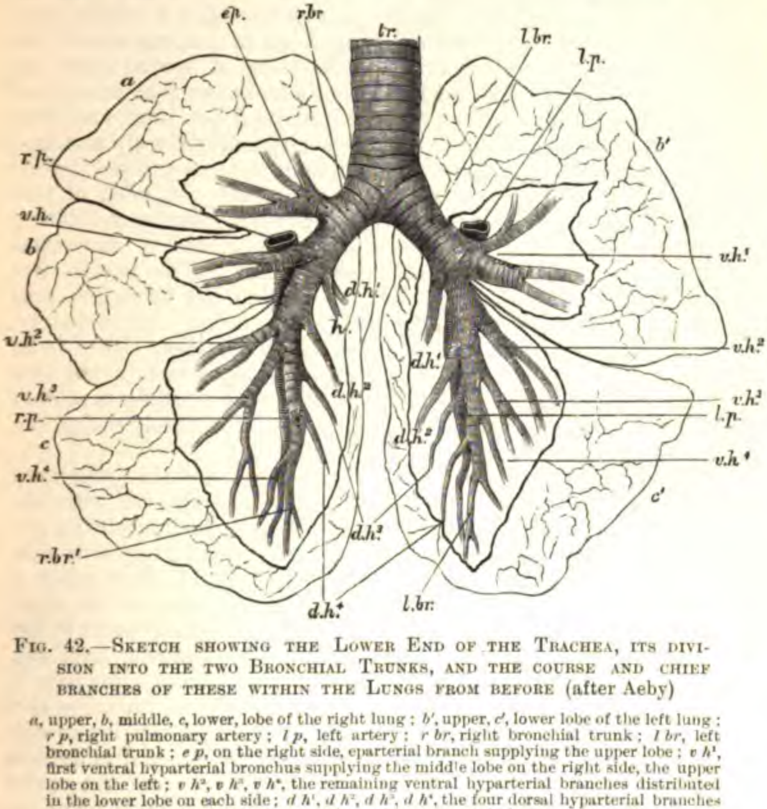


FIG. 42.—SKETCH SHOWING THE LOWER END OF THE TRACHEA, ITS DIVISION INTO THE TWO BRONCHIAL TRUNKS, AND THE COURSE AND CHIEF BRANCHES OF THESE WITHIN THE LUNGS FROM BEFORE (after Aeby)

a, upper, *b*, middle, *c*, lower, lobe of the right lung; *b'*, upper, *c'*, lower lobe of the left lung; *r p*, right pulmonary artery; *l p*, left artery; *r br*, right bronchial trunk; *l br*, left bronchial trunk; *e p*, on the right side, eparterial branch supplying the upper lobe; $v\ h^1$, first ventral hyparterial bronchus supplying the middle lobe on the right side, the upper lobe on the left; $v\ h^2$, $v\ h^3$, $v\ h^4$, the remaining ventral hyparterial branches distributed in the lower lobe on each side; $d\ h^1$, $d\ h^2$, $d\ h^3$, $d\ h^4$, the four dorsal hyparterial branches distributed on both sides in the posterior and inner part of the lower lobe; *h*, accessory bronchus arising close to the first dorsal hyparterial bronchus on the right side, and representing the one which supplies the azygos lobe in some animals. The main branches of the pulmonary vessels are distributed like the bronchi. Within the lung the arterial trunks run behind the bronchial branches, the venous trunks in front.

to the descent of the diaphragm the lower part of the chest cavity is extended downwards and inwards.[1]

The sectional area of the bronchial tubes, on the whole, increases as they subdivide. It has been stated above that the lateral branches are often quite as large as the main bronchus, so that the description here given, though most interesting and important from the point of view of comparative anatomy, is not of very great value from the practical aspect. It does not help the surgeon in his search for a foreign body of less than a certain size, and it has no great interest for the physician in helping him to the determination of the reasons for the selection of special spots for pulmonary cavities. Practically the surgeon knows that bronchi of very considerable size will be found throughout the lung, and that each of these bronchi will have a pulmonary artery closely attached to it on its posterior aspect, while pulmonary veins will not be far off on the anterior aspect, and he is often clearly reminded of this fact when, on puncturing the lung, the needle, after encountering some resistance, obviously enters a bronchus, and a somewhat copious hæmoptysis is the immediate result. The needle has passed through a large vessel before entering the bronchus, and hence the often somewhat alarming occurrence. Unfortunately, more often than not, he has to make the exploration either from the front or from the back, and cannot, therefore, avoid this accident, but, on the other hand, fortunately the bleeding is seldom serious unless the patient have been placed too deeply under the influence of the anæsthetic.

Pulmonary veins.—Within the lung itself the pulmonary veins (with the exception of the smallest) generally run with the bronchial tubes and arteries, the bronchial tube having on the one side the arterial branch, and on the other the vein, which is not, however so closely applied to it as is the artery, and often takes a somewhat different direction. The absolute position of the veins in relation to the bronchial branches varies in different parts of the lung, as may be seen from the accompanying drawing (fig. 43). On approaching the hilum, and in the root, the larger veins have a different arrangement, and run independently of the air tubes and the arteries. Thus, on both sides, the veins from the lower lobe issue from the lung behind and below the bronchus, and unite into the lower trunk (figs. 32 and 33); while the veins from the upper lobe on the left side, and from the upper and middle lobes on the right side, emerge in front of the air tube. and join together to form the upper trunk. Both trunks are very short, not exceeding three-quarters of an inch in length, and are directed nearly horizontally inwards and forwards on each side of the posterior part of the pericardium, which they pierce, and then immediately enter the left auricle.

---

[1] C. Hasse, 'Der Bau der Lunge des Menschen, bedingt durch die Bewegungen der Brustwandung bei der Atmung,' *Verhandl. des* 10. *Internat. Medic. Kongresses*, Berlin, 1890, Bd. ii. Abt. i. Anatomie ; 'Ueber den Bau der menschlichen Lungen,' *Archiv für Anat. u. Phys.* Anat. Abth. Jahrgang 1892.

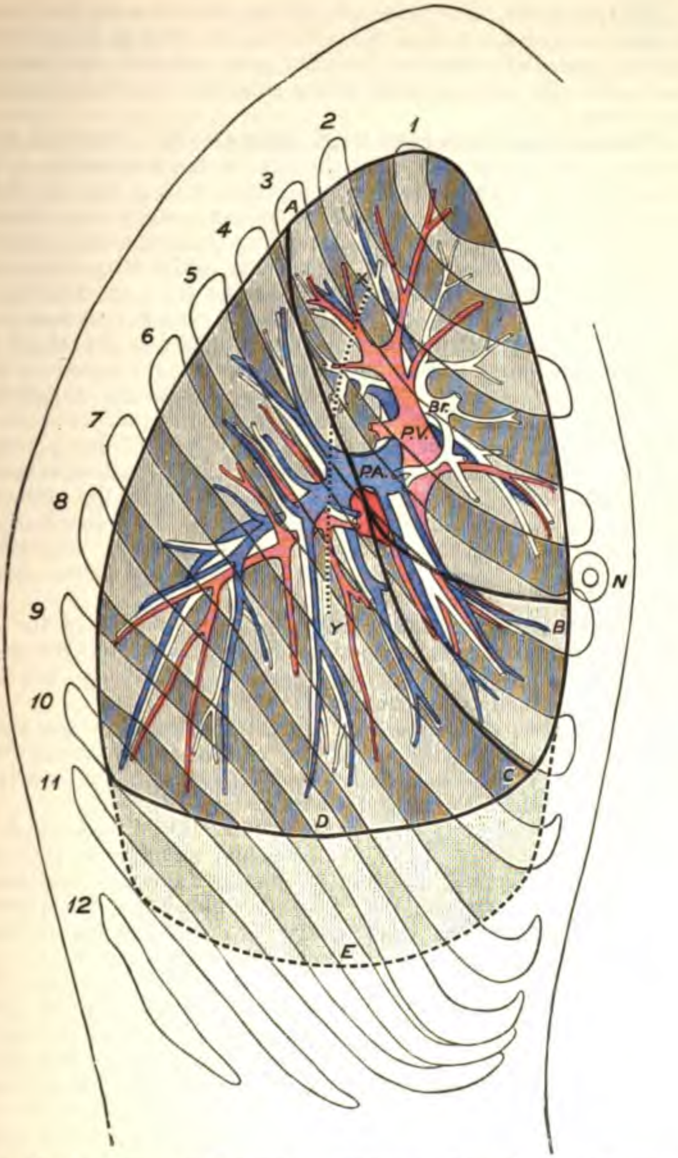


Fig. 43.—Dissection of Bronchi and Pulmonary Vessels from the Side

The subject was imbedded and the ribs were drawn before they were removed. The lobes of the lung were drawn before the final dissection was made. 1 to 12, ribs; *A B*, *A C*, fissures between upper lobe and middle and lower lobes; *D*, lower border of lung; *E*, lower limit of pleura; *Br*, bronchi; *P A*, pulmonary arteries; *P V*, pulmonary veins; *N*, nipple; *X Y*, front of vertebral bodies.

CHAPTER II

PHYSICAL DIAGNOSIS

PAGE
- Regions of the chest . . . 50
- Examination of the chest . . . 51
- Inspection . . . 51
- Palpation . . . 53
  - Fremitus—Vocal . . . 53
    - Rhonchal . . . 53
    - Friction . . . 53
    - Tussive . . . 54
  - Position of the cardiac impulse . . . 54
- Percussion . . . 54
  - Theory of percussion sounds . . . 55
  - Normal percussion sounds . . . 56
  - Morbid " " . . . 57
    - Increased resonance . . . 57
    - Tympanitic " . . . 57
    - Skodaic " . . . 57
    - Diminished " . . . 57
    - Absence of " . . . 57
      - Dulness . . . 57
      - Tubular percussion note . . . 57
      - Cracked-pot sound . . . 57
- Auscultation . . . 58
  - Breath sounds . . . 58
  - Varieties of respiratory sounds . . . 58
  - Adventitious sounds . . . 61
    - Varieties . . . 61
  - Vocal resonance . . . 65
    - Varieties . . . 65
- Mensuration . . . 66
- Cardio-pulmonary sounds . . . 67

THE presence of disease is indicated by the occurrence of symptoms which are, strictly speaking, subjective sensations of the patient, and the presence of signs which are phenomena recognisable by an observer.

Signs discovered by the aid of the various methods employed in diagnosis are termed 'physical signs.'

REGIONS OF THE CHEST

For convenience of description, the chest is divided into certain regions, but, as the terminology is somewhat variable, it is best in all cases to indicate the position of any physical sign by reference to certain vertical lines and a given rib or interspace.

The vertical lines are as follows: (1) The mid-sternal line; (2) the nipple line; (3) the anterior, mid, and posterior axillary lines; (4) the posterior scapular line (vertebral border of the scapula); (5) the vertebral line. Other terms are used by some writers, but they are now rarely employed in practice—*e.g.* the 'parasternal line,' midway between the margin of the sternum and the nipple line. The normal position of the cardiac apex is generally thus described: 'In the fifth left interspace one inch within the nipple line.' It is, however, preferable, in recording exact observations on the position of the cardiac impulse, to measure the distance from the mid-sternal line.

The following regions are commonly spoken of without their boundaries being accurately defined:—

Anteriorly.—The supra-clavicular, the infra- or sub-clavicular, the mammary, and infra- or sub-mammary regions.

The supra-sternal notch, the upper and lower sternal regions.

Laterally.—The axillary and infra-axillary regions, or upper and lower axillary.

Posteriorly.—The supra-spinous region or fossa, the infra-spinous region, the interscapular region, and the base.

EXAMINATION OF THE CHEST

The examination of the chest is conducted in the following order: (1) inspection, (2) palpation, (3) percussion, (4) auscultation, (5) mensuration.

It may be confidently observed that, no matter how experienced the examiner, it is rarely advisable to depart from this order, as so doing may entail the necessity of recommencing the examination when it is half finished.

It is essential in every case to examine the whole of the chest, and not merely some part of it which is thought to be the most probable site of disease.

INSPECTION

This preliminary step, although much neglected by students, is certainly in importance equal to, if not greater than, any other part of the examination. So much is there to be learnt from inspection, that it is often possible by simply looking at the chest to make a near approach to an accurate diagnosis.

We shall not attempt either to enumerate or describe all the morbid conditions which may be observed on inspection of the chest, as to do so would entail the repetition of much that may with greater advantage be considered elsewhere in this work. It will be sufficient to mention here the chief points to which attention should be directed in this part of the examination.

The patient should be placed in a good light, and the observer should stand opposite to him. The condition of the integuments and of the superficial vessels should be first noticed. The chest wall may be œdematous, or the veins enlarged. The *shape* of the chest is of importance. The normal chest is symmetrical and duly proportioned in its various diameters; the sternal angle (angulus Ludovici) at the junction of the manubrium with the body of the sternum, at the level of the second costal cartilages, is fairly marked.

In some persons the chest is long and narrow, the clavicles prominent and curved, the upper interspaces wide, and the antero-posterior and lateral diameters diminished, the shoulders sloping, and the scapulæ winged ('alar' thorax). In others in whom the chest is fairly broad, it is flattened in front.

The sternum may be prominent, as in the typical 'pigeon breast,' or it may be bordered on either side by the cartilages of the ribs, which are curved forwards to such an extent that the sternum lies in a hollow.

In children the subjects of rickets there is often a depression running obliquely downwards and outwards to the costal margin, and the ribs are 'beaded.' The 'barrel-shaped' chest typical of the large-lunged variety of emphysema may be noticed, with its prominent sternal angle and wide epigastric angle; or the small chest of the atrophic form of that disease. The point of the shoulder may be lower and the supra-clavicular fossa deeper on one side than on the other, and there may be a depression beneath the clavicle—signs indicative of shrinkage of the upper part of the corresponding lung.

Bulging of one side of the chest may indicate the presence of a pleural effusion, or a local bulging may be due to the presence of an aneurysm of the aorta.

Retraction of some part of the chest wall may have resulted from previous disease of the lung or pleura.

The heart may be uncovered by the retraction of the left upper lobe, and the impulse of the heart may be displaced from a variety of causes.

On the posterior aspect of the chest it is important to notice the distance of the border of the scapula from the spines of the vertebræ, and to observe whether the angles of the scapulæ are at corresponding levels on the two sides. The presence of a curvature of the spine should be looked for, and any undue obliquity of the ribs and narrowing of the intercostal spaces noted.

The expansion of the normal chest is equal on the two sides, and causes an increase in all its diameters. In disease the expansion may, from various causes, be unequal on the two sides, or enlargement may be effected by the descent of the diaphragm, the chest only moving upwards *en masse*, or the chest may be rigid and motionless on inspiration. Collapse of a sufficiently extensive area of a lung is accompanied by absence of expansion of the corresponding part of the chest wall.

Obstruction to the entrance of air into the lungs is indicated by recession during inspiration of the supra-clavicular fossæ, the suprasternal notch, the intercostal spaces, and the epigastrium.

In children the thoracic walls are yielding, and obstruction to the entrance of air may be accompanied by marked recession of the lower lateral parts of the chest wall.

PALPATION

Palpation is the use of the hands in physical examination, in order to obtain information through the medium of the sense of touch.

Fremitus.—VOCAL FREMITUS is the vibratory sensation conveyed to the hand when applied to the chest of a person during phonation.

The nature of the sensation perceived will depend upon the character of the voice, there being wide differences in this respect. The deep vibrations of a man's voice are always much better conducted than the higher-pitched voice of a woman or child. The amount and kind of the tissues interposed between the hand and the lung will also influence the vocal fremitus, which is usually most marked in lean persons. Any condition which produces consolidation of the lung will increase the vocal fremitus, provided the bronchi are patent; whilst the presence of fluid or air between the lung and the chest wall will diminish it, or altogether prevent its being felt.

The vocal fremitus is normally more distinct beneath the right clavicle than on the opposite side. If, therefore, the vocal fremitus is equal on the two sides and well marked, it is probably increased at the left apex, and a presumption arises that the left upper lobe is the seat of disease. If the fremitus is more marked at the left apex than the right, the presumption that the left upper lobe is affected is still stronger. If, on the other hand, the vocal fremitus beneath the clavicles is equal, but less marked than normal, probably the voice conduction is diminished at the right apex, and the condition of the right upper lobe must be carefully investigated in the further course of the examination. The significance of alterations of the vocal fremitus in the supraspinous fossæ is the same as corresponding changes beneath the clavicles.

Similar indications may be obtained in children from the fremitus felt during the act of crying.

RHONCHAL FREMITUS is felt when the vibrations produced by the passage of air through a bronchial tube, narrowed by the presence of mucus, are sufficiently intense to reach the hand applied to the chest. It is often present in bronchitis, and is especially frequent in the case of children suffering from that disease. When from any cause the breathing is accompanied by 'stridor' the vibrations may be also conducted to the chest wall.

FRICTION FREMITUS.—The vibrations produced by the rubbing together of two inflamed surfaces of the pleura, and more rarely of the pericardium or peritoneum, may sometimes be appreciated by the hand. The above term is used to indicate this sign. It is not uncommonly present in cases of dry pleurisy.

TUSSIVE FREMITUS is felt when the hand is placed upon the chest whilst the patient coughs. It is sometimes of use in diagnosis in the case of children, and in adults when the condition of the voice prevents the production of vocal fremitus.

Crepitus due to fracture of a rib, the fine crackling sensation felt in subcutaneous emphysema, and crackling due to the presence of blood beneath the skin may also be discovered on palpation.

The position of the cardiac impulse, which may or may not have already been observed on inspection, will be more definitely ascertained by palpation. It may be here stated that the importance in all cases of fixing this point accurately cannot be over-estimated, as *the position of the cardiac impulse is the key to the diagnosis of many affections of the chest.*

The presence of *pulsation* in any abnormal bulging will be noted, and *tenderness* may be complained of on palpation. When due to hyperæsthesia of the surface the slightest touch may cause intense suffering, whereas if the patient's attention is distracted by conversation, deep pressure may be made without so much as attracting attention to the spot.

PERCUSSION

Percussion is a method of physical examination in which some part of the body is struck with a view either to elicit sound or to estimate density of substance.

A slight difference in resonance brought out by an experienced examiner can be easily appreciated by others, but it requires long practice before the student is able to elicit the true note for himself, percussion being by far the most difficult of the arts of physical diagnosis. As an indication of the condition of the subjacent structures, the degree of 'resistance' experienced on percussion is only second in importance to the alteration in sound.

Method of percussing.—The chest may be struck directly with an instrument or the fingers (the *plessor*)=immediate percussion, or the blow may be delivered upon some intervening substance (the *pleximeter*)=mediate percussion. The use of any instrument, either in the form of a hammer as a plessor or a flat or other shaped piece of ivory as a pleximeter, was, in this country at least, until lately almost a thing of the past. Recently various new forms of pleximeter have been invented, and some of them are used by physicians of experience. It is, however, essential that the student should begin by learning to use his fingers, whatever he may do at a later period. The use of the finger has additional advantages, in that it enables one to appreciate any increase of resistance, and to feel the vibrations of the chest wall.

The middle finger of the left hand is generally used. It must be applied firmly to the part, and should be struck with one, two, or three fingers of the right hand, the fingers being flexed, the blow delivered from the wrist and not from the elbow, and vertically to the pleximeter.

Light percussion is best when it is desired to elicit sounds from the parts immediately subjacent, or to map the outline of the thoracic or abdominal viscera, also whenever there is tenderness of the chest wall, and, speaking generally, when examining the front of the chest. Over the supraspinous fossæ and interscapular regions, it is often necessary to give a firm blow in order to bring out slight differences in resonance. This is sometimes termed 'deep' percussion.

The clavicles are percussed by tapping them directly with the finger. It is often possible to make out slight differences in resonance by percussion of the clavicles when no variation from the normal can be detected elsewhere; also if the chest is percussed after a deep inspiration a slight difference in resonance, due to diminished entry of air into an affected part of the lung, may be discovered. These steps should therefore never be omitted in the examination of doubtful cases of tuberculosis of the apex of the lung.

The sounds elicited from different chests and from different parts of the chest vary considerably; it is therefore essential in all cases to compare the note obtained over any spot with that over the corresponding part on the opposite side; interspace must be compared with interspace, and rib with rib, the finger being similarly applied and struck with equal force on each side.

In percussing the back of the chest the patient must be directed to lean forwards, to fold the arms, and to 'let the shoulders drop,' so as to completely relax the scapular muscles.

Auscultatory percussion is a term applied to the combined use of percussion and auscultation. The method is of service in defining the outlines of a large hollow cavity, such as a dilated stomach, a difference in the note being appreciated through the stethoscope when the pleximeter passes the boundary of the cavity. At the present time great importance is attached by some writers to the use of this method of diagnosis in determining the outlines of the heart.

Theory of percussion sounds.—As already stated, percussion is an art only to be acquired by long practice, and its acquisition and application are but little assisted by a knowledge of the various conflicting theories which have been from time to time put forward to account for the normal and abnormal sounds so elicited.

It is now accepted by most writers that the sound which is obtained when the healthy chest is percussed is 'mainly due to the vibration of the thoracic walls alone' (Bristowe), and that any condition which interferes with the vibration of the chest walls alters the percussion note. According to another view, however, the middle-sized and largest bronchi (Gee) are the seat of the vibrations which produce the normal percussion tone.

The nomenclature of percussion sounds is almost as confusing as that of the breath sounds. The terms mentioned in the Provisional Report of the Committee of the International Medical Congress (1881) have been here adopted.

Before proceeding to describe them it is necessary to refer to some of the synonyms in ordinary use, and also to describe the normal percussion signs. The term 'dulness' is held to imply absence of 'tone' (Gee), 'tone' being the result of the regular succession of impulses which are present in a musical sound, but lacking in a mere noise.

Of percussion sounds the highest in pitch is that produced by percussion over a bone (osteal); next in descending scale comes a note resembling that produced by percussion of the trachea when the glottis is open (tubular or tracheal); the lowest being that produced by percussion over a hollow cavity containing air, *e.g.* the stomach (tympanitic). The note over healthy lung (normal percussion tone) comes in point of pitch after the tracheal tone.

Normal percussion sounds.—By long practice it is possible to form a rough standard of resonance for different regions of the chest, regard being had to the condition of the parietes and other considerations. This is of service when the percussion note of healthy lung cannot be obtained owing to the presence of disease in corresponding areas on both sides of the chest.

Taking the front of the chest from above downwards, the normal condition of the percussion sounds is as follows:—

Resonance commences about 1½ inch above the clavicle; it is equal on the two sides until the sound is modified owing to the presence of the liver on the right side, and of the uncovered portion of the heart on the left. The whole of the sternum is resonant in the normal condition.

The *liver dulness* commences at the upper border of the sixth rib in the nipple line, and extends to the costal margin. A horizontal line drawn outwards on a level with the base of the ensiform cartilage crossing the eighth rib in the mid axillary line and the upper border of the eleventh rib posteriorly marks its upper border. In the median line the liver dulness extends about 1½ inch below the base of the ensiform cartilage.

Precordial dulness.—The area of superficial precordial dulness, as obtained on light percussion, is bounded above by the fourth left costal cartilage, on the right by a line drawn along the left edge of the sternum, on the left by a vertical line drawn through the fifth left intercostal space an inch within the nipple line (normal site of the apex beat), and below by a horizontal line from the apex beat to the sternum.

It is usually stated to merge below into the liver dulness, although it is said to be possible to differentiate the two.

Behind.—The percussion note in the supraspinous fossæ, owing to the greater thickness of the muscles, is less resonant. In the interscapular region the resonance increases somewhat, and in the infrascapular region the note is almost equal to that beneath the clavicles.

On the right side resonance extends to the tenth rib, and on the left to the eleventh rib or thereabout.

Morbid percussion sounds.—The following varieties may be recognised:—

INCREASED RESONANCE: HYPER-RESONANCE.—This sound has the quality of the normal percussion note to an exaggerated degree. It is of more marked intensity than the normal note and of lower pitch. The percussion note over an emphysematous or over-distended lung may be of this character.

TYMPANITIC RESONANCE.—A somewhat musical sound of varying pitch heard on percussion over a distended stomach or intestine, also over a pleural cavity containing air (pneumothorax), over a very large pulmonary cavity, and in complete excavation of the lung.

SKODAIC RESONANCE.—A clear sound of higher pitch than the last variety, but of the same quality, produced on percussion over the upper part of the chest, particularly the infraclavicular region, when the pleural cavity contains a certain amount of fluid, and also in some cases of pneumonia of the lower lobe.

A similar note may occasionally be elicited by percussion over an area of deep-seated or incomplete consolidation of the lung.

When present in cases of pleural effusion skodaic resonance beneath the clavicle is believed to be produced by relaxation of the upper part of the lung, which, however, still remains in contact with the chest wall. Dr. Bristowe attributes the raised pitch of this sound to the diminution in the vibrating area present under such conditions.

DIMINISHED RESONANCE (*Dulness varying in degree*).—A sound shorter, sharper, and higher in pitch, and lacking the tone of the normal note. It may be of a quality similar to that produced by percussion over a piece of wood (wooden) or a bone (osteal). The sound usually signifies either incomplete consolidation or displacement of the lung, or the presence of some morbid condition of the pleura or chest wall. With diminished resonance there is almost always increased resistance on percussion.

ABSENCE OF RESONANCE (*Dulness*).—A high-pitched note from which tone is more or less completely absent. The sound elicited by percussion over a pleural cavity containing fluid may be absolutely dull. Over consolidated lung the loss of tone is not usually complete.

Tubular Percussion Note and Amphoric Resonance.—A sound similar to that obtained by percussing the trachea with the glottis open. It is usually produced over a large superficial cavity with free bronchial communication. This may be accompanied by the following

Cracked-pot Sound (*Bruit-de-pot-fêlé*).—A sound having an amphoric and slightly metallic quality produced on percussion over a superficial cavity, with slightly yielding walls and free bronchial communication, the mouth being at the time open.

AUSCULTATION

Auscultation may be practised directly, by applying the ear to the chest wall, or mediately, by means of a stethoscope. The forms of stethoscope in common use are the rigid tube of wood, vulcanite, or metal, with broad ear-piece and narrow chest-piece, and the flexible, single or binaural, instrument. The first has the advantages of enabling the auscultator to appreciate any abnormal impulse of the heart or of an aneurysm, and also that it can be applied to the chest when covered with a layer of clothing, whereas the flexible instrument must be applied accurately to the skin itself. The binaural stethoscope, however, is more convenient for the examination of children and of patients in the recumbent position.

Breath sounds.—It is a misfortune to medicine, and a source of great difficulty to students, that the nomenclature of the auscultatory sounds should be in such a state of confusion. As uniformity in this respect can only be obtained by sinking individual preferences and submitting to the dictates of authority, it would have been a great advantage if the terminology recommended by the majority of contributors to the Provisional Report of the Committee of the International Medical Congress had been generally adopted. Unfortunately this has not been the case, and matters remain much as they were years ago.

A knowledge of the normal breath sounds is obviously an essential preliminary to the auscultation of the chest in disease, and this can only be obtained by the examination of a large number of healthy subjects.

Three varieties of respiratory sounds are to be recognised in every healthy chest—(1) the sound produced at the glottis, and audible over the lower end of the trachea, which is termed 'tracheal' or 'bronchial' breathing; (2) the sound which is believed by some writers to be produced in the alveoli and which is audible over the lungs, termed 'vesicular' breathing; (3) the sound audible about the roots of the lungs, which combines the characters of the two preceding, and is termed 'broncho-vesicular' breathing. As the normal respiratory sounds vary considerably in intensity in different individuals, there is no common standard of reference, and one side of the chest must always be compared with the other. Any change in the *quality* of the sounds, however, is to be regarded as abnormal.

Varieties of respiratory sounds.—VESICULAR BREATHING.—This is a soft rustling sound audible during inspiration. It is generally followed, without any appreciable interval, by a sound, of lower pitch, shorter duration, and faintly blowing quality, heard during expiration, but this may be absent. The two sounds constitute the normal respiratory murmur audible over the healthy lung.

Prolonged Expiration.—The expiratory sound may be prolonged—a sign the significance of which varies with the pitch and quality of the sound. If high pitched and bronchial or tubular in quality, it usually indicates consolidation or compression of the lung; if, on the other hand, the pitch is low and the quality faintly blowing, but not bronchial or tubular, emphysema is probably the cause.

Exaggerated Breathing.—This is a sound possessing the characters of vesicular breathing in an exaggerated degree. It is the normal breath sound of children, but when met with in adults indicates that an increase of function in one part is compensating for deficient action elsewhere.

Exaggerated breathing is perhaps best heard over the unaffected side in cases of pleural effusion.

Diminished Breathing.—The characters of the normal breath sound are retained, but the intensity is much diminished. It is present under a variety of conditions involving lessened functional activity of the lung, and very commonly at the apex in an early but not the first stage of tubercular disease.

Suppressed Breathing.—The breath sound is absent, a condition met with in some cases of pleurisy with effusion, also in certain cases of pneumonia, when the bronchi are filled with exudation (massive pneumonia), in collapse of the lungs, and when from any cause a bronchus is completely occluded.

Interrupted Breathing; Wavy or Cogwheel Breathing.—The inspiratory sound is divided into two or three parts, owing to an irregular expansion of the lung, the result of disease of the bronchi or of the lung interrupting the entrance of air. It may also be due to irregular contraction of the muscles of respiration, and possibly also to the presence of pleural adhesions. It may be heard at the apex of the lung in the earliest stage of tuberculosis, but, taken alone, is an untrustworthy sign owing to its close simulation by the breath sound present in certain nervous conditions. It is also occasionally simulated by a succession of 'air-wave' murmurs (cardio-pulmonary) produced by the impact of the heart upon the lung.

Bronchial Breathing is the glottic sound, as heard in the normal chest over the seventh cervical spine. The sound there audible may be taken as the standard of comparison by which to gauge the quality of similar sounds heard elsewhere in the chest of the same patient, the term 'bronchial breathing' being used to signify that the sound heard over an area of disease in the lung is of the same quality as that audible over the seventh cervical spine. It is of variable intensity, and differs from vesicular breathing in that the inspiratory sound is higher in pitch and tracheal in quality, whilst the expiratory sound is higher in pitch, separated from the former by a distinct interval, tracheal in quality, usually more intense, and of equal or longer duration.

When heard in pneumonia or tuberculosis, it signifies the presence of consolidation in an area of lung. In large pleuritic effusions it may be audible over the site occupied by the lung, and indicates that it is compressed or collapsed. It is occasionally audible over the whole of the affected side in cases of pleural effusion, especially in children; when this occurs in adults, it is considered by some writers to indicate the existence of positive intrathoracic pressure; as, however, it is occasionally present in cases of moderate effusion, that statement cannot be absolutely accepted. It probably indicates that the large bronchi of the collapsed lung are still patent. When a thoracic aneurysm or mediastinal growth is situated between the trachea or a large bronchus and the chest wall, bronchial breathing is often audible over the area occupied by the tumour.

Broncho-vesicular Breathing, as the name implies, combines the characters of bronchial breathing with those of the normal respiratory sound, and indicates the presence of an amount of consolidation short of that necessary to produce bronchial breathing. It is heard normally over the manubrium in front, and the upper part of the interscapular region behind. In the inspiratory sound the vesicular quality is diminished, but not absent; the tracheal quality is more or less marked, according to the degree of consolidation; the pitch is raised in proportion as the tracheal quality predominates over the vesicular; the intensity is variable.

The pitch, tracheal quality, and length of the expiratory sound correspond with the characters of the inspiratory sound.

Tubular Breathing is the glottic sound conducted through an area of lung completely consolidated.

Tubular breathing and bronchial breathing are often used as synonymous terms, but the former sound differs from the latter in possessing a ‘whiffing’ character, and in its higher pitch.

It is present typically in pneumonia, when the bronchi are patent. If present in tuberculosis, it indicates that a portion of lung situated near the surface is *completely* consolidated, the bronchi being patent.

Cavernous Breathing is a modification of the glottic sound, produced by the presence of a cavity within the lung or external to it, in communication with an open bronchus.

The inspiratory sound is low-pitched, hollow, and blowing; the expiratory sound is still lower in pitch, has the hollow, blowing character more marked, and is usually more prolonged.

It requires a partially empty cavity, in communication with an open bronchus, and at least equal in size to an unshelled walnut, to produce a breath sound of this character.

The breath sound audible over the supposed cavity should always be compared with that over the lower end of the trachea, as errors in the diagnosis of cavities are of frequent occurrence, owing to the neglect of this precaution.

Amphoric Breathing is a sound of variable intensity presenting the hollow, blowing character of cavernous breathing in an exaggerated degree, and with the addition of a distinctly ‘metallic’ quality.

It indicates the presence of a large cavity, either in the lung or in the pleura (pneumothorax), in communication with an open bronchus.

The difference between vesicular, bronchial, tubular, cavernous, and amphoric breathing may be expressed in tabular form. Thus:

—	Pitch	Quality	Interval	Duration	Intensity
			Vesicular Breathing		
I.	Low	Vesicular	None	E. shorter than I.; or the sound may be absent during E.	Variable
E.	Lower	Blowing			—
			Bronchial Breathing		
I.	High	Tracheal (seventh cervical spine)	Distinct	E. equal to or longer than I.	Variable
E.	Higher	Do.			Greater
			Tubular Breathing		
I.	Higher than in bronchial breathing	Laryngeal or whiffing	Distinct	E. equal to or longer than I.	Variable
E.	Higher	Do.			Greater
			Cavernous Breathing		
I.	Low	Blowing and hollow	Distinct	E. longer than I.	Variable
E.	Lower	Both characters more marked			Greater
			Amphoric Breathing		
I.	Low	Hollow and metallic	Distinct	E. longer than I.	Variable
E.	Lower	Both characters more marked			Greater

I. = inspiration; E. = expiration.

Adventitious sounds.—A great variety of sounds to which, as a whole, the above term is applied, become audible in diseased conditions of the lungs, bronchi, and pleuræ.

FRICTION SOUND is the name given to the rubbing, crepitant, or creaking sound heard on auscultation over the large serous sacs when their ordinarily smooth surfaces are roughened, usually by an inflammatory exudation.

In the case of *the pleura*, apart from the rubbing nature of the sound, its evidently superficial character, and the fact that it is usually audible both with inspiration and expiration, help the diagnosis. It is, however, often very difficult to decide whether a sound having a crepitant character is of pleural or pulmonary origin. In quiet breathing, if the sound is audible from the very beginning of inspiration, or if it only appears at the end of a deep inspiration, if it remains unchanged by cough, and if there is localised pain increased by inspiration, it is probably due to the presence of a patch of exudation on the underlying pleura, or of fine fibrous adhesions. The pleural origin of a crepitant sound is often indicated by the discovery of a 'rubbing' friction sound in its immediate neighbourhood.

A pleural friction sound in the precordial area may acquire a 'to and fro' character and cardiac rhythm under the influence of the movements of the heart. Such a sound usually ceases when the lung is fixed by holding the breath after a deep inspiration.

RHONCHI are dry, musical sounds produced in the bronchial tubes. When low-pitched, loud, and snoring in character, they are termed *sonorous*; when high-pitched and whistling, they are called *sibilant*, the pitch of the sound being chiefly dependent upon the calibre of the tube in which it is produced. Sonorous rhonchi usually indicate that mucus is present in the large tubes; sibilant rhonchi that the lining membrane of the smaller bronchi is swollen. The former often disappear when the patient coughs; the latter are usually uninfluenced by cough.

Rhonchi may be audible during either inspiration or expiration, or may accompany both parts of the respiratory act; in bronchitis and asthma they are, as a rule, especially marked during expiration.

STRIDOR is a harsh vibrating sound resulting from obstruction of the larynx, trachea, or a main bronchus. This sound may be caused by a variety of local conditions, but is in thoracic disease most often due to direct pressure on or narrowing of the trachea by an aneurysm or an intrathoracic tumour, or to syphilitic stenosis of the trachea, or to bilateral paralysis of the abductors of the vocal cords, the result of pressure on one pneumogastric nerve or on both recurrent laryngeal nerves.

RÂLES are sounds produced by the passage of air through exudation in the alveoli or bronchi, and by the separation of the surfaces of collapsed alveoli or of the swollen lining membrane of the finer bronchi. They vary much in character, being chiefly influenced by the condition of the lung, in the neighbourhood of which they are produced. The presence of consolidated lung gives a sharp, crackling, explosive quality to the sound, whereas, when the surrounding lung is of normal texture, it is usually of a bubbling character. Sounds of the first variety are termed (1) small, (2) medium, or (3) large, crackling or crepitant râles.

(1) *Small crackling râles* are sharply defined crackling sounds of small size, and often few in number, chiefly audible during inspiration, but also during expiration.

They indicate the presence of a lesion in the ultimate divisions of the bronchi, or in the pulmonary alveoli, the surrounding lung being usually in process of consolidation. Sounds of this character are present when a tubercular deposit is forming, or a pneumonic exudation is undergoing softening.

(2) *Medium crackling râles* are sounds presenting the same general characters as the above, but of larger size. They occur during the softening of tubercular deposits, also in broncho-pneumonia, and in the process of absorption of a pneumonic exudation.

(3) *Large crackling râles* are sounds produced at the site of softening in the lung, when the process has extended to such a degree that small cavities, the size, perhaps, of a pea or hazel-nut, have formed, the surrounding lung being still consolidated. They are usually fewer in number than either of the former varieties. Râles of this character are of frequent occurrence in tuberculosis, and are also heard when a pneumonic area is breaking down.

Adventitious sounds, presenting a sharp ringing or metallic character, are sometimes called '*consonating râles*.' The term is, however, founded upon a misconception, and it would be an advantage if it were no longer employed.

Moist sounds produced in the neighbourhood of spongy lung are termed (1) small, (2) medium, and (3) large bubbling or mucous râles.

(1) *Small bubbling râles* are sounds differing from the corresponding râle of the former variety in that they are less sharply defined, and suggest the bursting of a soft bubble rather than the explosion of a minute shell. They are produced by the passage of air through fluid in the bronchioles, the surrounding lung being of spongy texture. Sounds of this character are heard in cases of capillary bronchitis, especially in children.

The term 'subcrepitant' is sometimes applied to sounds of this character when the crackling quality is present, but to a diminished degree.

(2) *Medium bubbling râles.*—These sounds are similar in quality to those above described, but are produced in larger tubes, and are therefore of larger size.

(3) *Large bubbling râles.*—The character of these sounds is almost sufficiently indicated by the name. They are produced in the larger bronchi and trachea by the passage of air through frothy mucus, and are heard in cases of bronchitis and pulmonary engorgement when the vital powers are failing.

Gurgling râles are large, liquid sounds of a reverberating character; they are produced in a cavity in the lung, and are best elicited by making the patient cough.

Clicking sounds.—The character of these sounds is best illustrated by whispering the word 'click.' They differ from râles in being more 'sticky' in quality,' are produced during inspiration only, and are most often heard at the apex of the lung in tuberculosis. The presence of 'clicks,' as they are called by those w make use of the term, is generally considered to indicate the commencement of softening in a tubercular deposit.

These sounds are not distinguished by German writers from crackling râles, and it must be admitted that the difference is but slight, and with difficulty appreciated by students of auscultation.

Crepitation is a sound almost exactly similar to that produced by rubbing the hair between the fingers close to the ear. It requires no qualifying adjective, such, for example, as 'fine,' and its use should be strictly limited to sounds of this character.

Crepitation is caused by the passage of air into vesicles either containing a fibrinous exudation or in a condition of collapse.

It is heard typically during the latter part of inspiration in the early stage of pneumonia, and also at the bases of the lungs when in a state of œdema, lobular collapse, and hypostatic pneumonia.

Metallic tinkling and Amphoric echo.—When a sound is produced in or near a large dense walled cavity (pulmonary or pleural) containing air, or in a neighbouring bronchus in communication with it, the audible echo of the sound acquires a peculiar quality, which, when single, clear ringing and high pitched, is best described by the terms 'metallic' and 'tinkling.' When low pitched and of 'buzzing' rather than 'ringing' quality it is termed amphoric echo. The 'metallic' quality of the sound is also present in amphoric echo but is less marked. Sounds of this nature may be produced during the act of breathing, by coughing, by speaking, or by the movement of the heart.

Succussion splash.—A peculiar splashing sound, described by Hippocrates, is produced by the sudden movement of air and fluid in a cavity. It is present in cases of hydro- or pyopneumothorax, and is elicited by shaking the patient or making him cough. In cases of thoracic disease it must not be confounded with a similar sound originating in the stomach.

Bell sound.—In pneumothorax, and when a very large cavity is present in the lung, if a coin placed flat upon the chest is struck with another coin, a sound like the tinkling of a bell may be audible through the stethoscope applied over the affected area.

Post-tussive suction is, as its name implies, a sound of 'sucking' character heard immediately after a cough. It is a very important and most trustworthy sign of a cavity in the lung. For its production it is necessary that the walls of the cavity should be yielding, so that during the act of coughing it may be compressed, when a slightly hissing sound becomes audible. By its re-expansion air is drawn rapidly through the bronchial orifice, and thus the sucking sound is produced. It is sometimes called 'the india-rubber ball sound.'

Veiled puff.—This sound, as described by Skoda, although the term was previously applied by Laennec to a sign the exact nature of which is doubtful, is a single, rather high-pitched sound of a 'puffing' character which becomes suddenly audible towards the end of inspiration. It suggests the impression that a whiff or puff of air has been blown into a small cavity situated immediately beneath the end of the stethoscope. It is, we believe, a valuable sign of the presence of sacculated bronchiectasia of small size.

Vocal resonance.—The voice sound, as heard over the healthy lung, is diffused, distant, and low pitched. It varies in intensity in different individuals, being chiefly dependent upon the quality of the voice. The vocal resonance is normally more intense in the neighbourhood of the trachea and large bronchi than elsewhere, and in the right infraclavicular region than in the left. It is *diminished* in intensity by the intervention of any badly conducting medium between the lung and the chest wall, a condition present in pleurisy with effusion, in some cases of thickening of the pleura, and in pneumothorax. It is also diminished owing to the extremely spongy condition of the lung present in emphysema. Occlusion of the bronchi, owing to the presence of morbid growths in the lung or mediastinum, or of solid fibrinous exudations filling the tubes, such as occasionally occurs in croupous pneumonia (massive pneumonia), produces a similar effect. The voice sound is *increased* in loudness, but without alteration in pitch, when a degree of consolidation of the lung is present insufficient to produce bronchophony, or when the voice is transmitted through a cavity not surrounded by consolidated lung (Flint). The conduction of the whispered voice is modified by the same conditions as affect the loud voice, and in a similar manner.

BRONCHOPHONY is a sound of variable intensity characterised by concentration of the transmitted voice, elevation of pitch, and a degree of nearness to the ear falling short of the condition requisite for pectoriloquy—*i.e.* the sound, though seeming to originate near to the end of the stethoscope, passes away from the observer, and does not appear to have been spoken from the end of the instrument straight into his ear. Bronchophony is usually found with bronchial breathing, and has the same significance. It is normally present over the lower cervical spines, in the upper part of the interscapular region, and over the sternal portion of the infraclavicular regions.

PECTORILOQUY is by some writers considered to be merely an exaggerated degree of bronchophony. It differs from it, however, in the fact that, whilst in the latter the *noise* only is transmitted, in pectoriloquy *articulate speech*, in addition to sound, becomes audible. Pectoriloquy has been divided into 'bronchophonic pectoriloquy' and 'cavernous pectoriloquy,' the one variety indicating the presence of consolidation of the lung, the other of a cavity. If pectoriloquy is accompanied by the characters of bronchophony (nearness to the ear and elevation of pitch) the transmission is by solidified lung; if, on the other hand, speech is transmitted, and the characters of bronchophony are wanting, the inference is that the pectoriloquy denotes a cavity (Flint). If words articulated in a whisper are conveyed to the ear of the auscultator, *whispering toriloquy* is said to be present. The sign has the same general nificance as pectoriloquy of the loud voice. Bacelli has sought prove that the character of a pleural effusion may be determined by the conduction or nonconduction of the whispered voice (*vide* p. 562).

Œgophony is a term applied to a modification of the vocal resonance, in which the transmitted sound has a nasal or bleating character. It occurs in pleurisy when the amount of effusion is moderate, and is most often heard about the angle of the scapula. It was believed by Dr. Stone to depend upon the interception of the fundamental vowel tone by the exudation and the passage of the harmonic overtones. For Dr. F. Taylor's views on the subject the chapter on 'Pleurisy' may be consulted (*vide* p. 561).

MENSURATION

Mensuration, or the act of measuring, is a means of physical diagnosis especially applicable in diseases and deformities of the chest.

The methodical examination of the chest concludes with mensuration, by which means the actual shape and dimensions of the chest as a whole, and the relative measurements of corresponding parts on the two sides, may be ascertained and recorded.

The instrument in general use for this purpose is the *cyrtometer*, which consists of two lengths of soft metal united by a short piece of india-rubber tubing.

The centre of the junction is applied to the vertebral spine at the desired level, and the metal pieces are accurately moulded to the chest wall, and are marked where they overlap in the midsternal line on a level with the point of application to the spine.

The instrument is now removed and placed upon a large sheet of paper, and a tracing taken of its outline. The antero-posterior, transverse, and other measurements are then determined.

In diseases of the lungs or pleura such records are chiefly of value in cases of suspected intrathoracic tumour or of pleurisy in the stage of effusion, or subsequently when collapse of the lung has occurred and treatment is being directed to promote its re-expansion; also in cases of tuberculosis in the stage of quiescence or arrest, when it is desired to ascertain the effect of climatic or other treatment upon the capacity of the chest.

It is necessary to bear in mind that the outline is that of the chest wall only, and that its shape will be altered by the development of muscle or the deposition of fat as well as by expansion of the lung, a source of fallacy which, to some extent, militates against conclusions derived chiefly from this method of examination.

By means of a tape the circumference of the chest can be determined, and differences on the two sides, either during expansion or in repose, can be determined by the use of single or double tape measures.

For the detection of subclavicular depression the trained eye is of more service than any instrument.

CARDIO-PULMONARY SOUNDS

Sounds closely resembling those resulting from organic disease of the valves of the heart, but really produced outside the heart, are frequently present in disease within the thorax. They are termed *false* or *cardio-pulmonary* murmurs.

The various conditions which may give rise to these sounds may be classified thus:—1. Displacement of, or pressure upon, the heart, the result of disease of (*a*) the lung, (*b*) the pleura, (*c*) the thorax, or (*d*) the abdomen. 2. Effusion into the pleural cavity. 3. Changes in the pleura of the præcordial area. 4. Changes in the lung overlying the heart. 5. Changes in the pericardium.

1. (*a*) Displacement of the Heart, the Result of Disease of the Lung.—As a result of the contraction of a cavity, say in the apex of the left lung, the heart is frequently found displaced upwards, or upwards and outwards. Under these circumstances a systolic murmur is often heard in the second or third left interspace, having its maximum intensity about two inches from the edge of the sternum. The presence of the cavity and of the indurated lung surrounding it intensifies the murmur, which may be so distinct as to be audible over the greater part of the left chest. This sound, though more commonly suggesting aortic disease or a hæmic murmur in the pulmonary artery, may be mistaken for the murmur of mitral regurgitation. When the presence of the cavity is detected, and the displacement of the heart recognised, the differential diagnosis should not be difficult, especially when the somewhat rare association of tuberculosis and valvular disease is borne in mind.

(*b*) Pressure upon or Displacement of the Heart, the Result of Disease of the Left Pleura.—Perhaps of all the false murmurs none are more common or present greater difficulties in diagnosis than those caused by pressure upon the heart resulting from the contraction of the left side of the chest after an attack of pleurisy. The pleura is probably thickened, and the lower lobe of the left lung partially collapsed, and at each systole a sound is produced by the sudden impact of the heart displacing the air in the larger bronchi. This may be audible not only at the apex and in the axilla, but also at the angle of the left scapula, in the trachea, and in the mouth.

In one case, seen by the writer, a systolic murmur produced in this manner was distinctly audible at a distance of two feet from the patient, and the presence of an aneurysm of the aorta compressing the trachea had been suspected. This murmur may be present even during attacks of functional palpitation.

(*c*) Displacement of the Heart from Deformity of the Chest.—Murmurs originating from this cause are occasionally met with. In a case under the care of the writer the lower part of the sternum was depressed to such an extreme degree that by careful measurement it was clear that its posterior surface was only separated from the spine by a distance of about two inches. The chest in section was shaped like a crescent with blunted horns, the left one holding the heart, and the organ could be, so to speak, grasped by the hand, and all its movements distinctly felt. In that case a loud systolic murmur was present.

(*d*) THE UPWARD PRESSURE OF A LARGE EFFUSION INTO THE PERITONEAL CAVITY is sometimes the cause of a false murmur, systolic in time, and audible at either the base or apex of the heart. When the fluid is withdrawn by paracentesis, the murmur disappears. A similar murmur has been heard in cases of hydatid of the liver.

2. EFFUSION INTO THE PLEURAL CAVITY.—DISPLACEMENT MURMUR.—In some cases of pleural effusion, more frequently when the left side of the chest is affected, a loud and prolonged systolic murmur may be heard, either near the apex or at the base of the displaced heart. Such a condition is likely to produce a murmur either in the pulmonary artery or in the aorta by pressure upon the vessel, or, what is more probable when the murmur is localised at the apex, within the ventricle itself. The murmur disappears on the removal of the fluid from the chest, in some cases immediately, in others not until some time after the operation.

3. CHANGES IN THE PLEURA OF THE PRÆCORDIAL AREA.—The sounds simulating cardiac murmurs resulting from this cause will be found described under 'FRICTION SOUNDS.'

4. CHANGES IN THE LUNG OVERLYING THE HEART.—In the subjects of pulmonary tuberculosis it is very common to hear a systolic murmur about the apex of the heart, which closely resembles the murmur of mitral regurgitation. The sound is caused by the impact of the heart upon the lung tissue partially consolidated, producing an audible sound by displacement of the air in the bronchi in a similar way to that already described. It is almost always most distinct during expiration, is superficial, high pitched, and disappears when the breath is held after a deep inspiration, and often becomes inaudible when the patient lies down. This murmur may sometimes be heard in the axilla and at the angle of the left scapula. The most common site of a murmur thus produced is not, however, at the apex of the heart, but in the second left intercostal space, about two inches from the sternum. In doubtful cases of early tuberculosis of the left upper lobe, the presence of this morbid sound is strongly suggestive of consolidation of the lung. In many of these cases, however, it is probably due to some displacement of the pulmonary artery, the result of the disease of the neighbouring lung.

Apart from disease of the lung, a false systolic murmur may often be heard about the cardiac apex in neurotic subjects when the action of the heart is markedly excited.

5. CHANGES IN THE PERICARDIUM.—False murmurs, when due to chronic changes in that membrane, are frequently heard at one particular spot—viz., the sixth left interspace and over the seventh rib close to the base of the ensiform cartilage. Here the

right ventricle is in contact with the chest wall, and it is on its anterior surface that the 'white patch,' due to localised thickening of the pericardium, is most commonly found. The sound produced by the movement upon each other of the pericardial surfaces at this spot is systolic in time, usually short, sharp, localised, and superficial; it seldom acquires a blowing character. In fact, it often resembles more nearly a rough reduplication of the first sound than a murmur. It is, perhaps, doubtful whether the altered sound, which is so commonly heard at this spot, is in all cases due to the presence of a 'white patch' on the pericardium. It is especially common in emphysema with downward displacement of the heart. The effect of change of position upon this sound is variable. It may disappear entirely when the patient is in the recumbent position, as often happens with friction sounds audible elsewhere over the heart, whilst at times it is hardly at all affected by such a change. When the heart is not displaced, a murmur presenting similar characters may often be heard in the fifth left interspace close to the sternum. Thickening of the pericardium of the left auricular appendix occasionally produces a rough systolic murmur in the second left interspace.

The following precautions should be observed in the examination of all cases presenting murmurs. Assuming that the presence or absence of those consecutive changes in the heart which are almost invariably associated with disease of any given valve has been noted, if there is still room for doubt, the following points must be observed carefully :—

1. The exact period in the cardiac cycle occupied by the murmur. False murmurs are often not exactly synchronous with the commencement of systole or diastole; they may precede or follow the one or the other.

2. The site of maximum intensity and the line of conduction of the murmur. These do not usually coincide with what is found with similar murmurs of organic origin.

3. The condition of the lungs. With doubtful apex murmurs evidence of pulmonary engorgement is strongly in favour of organic valve disease, as that condition is almost invariably absent in the reflux of anæmia.

4. The effect of change of position upon the sound. *It is essential to examine every case of suspected valve disease both in the standing and recumbent position.* It may be stated with confidence that an opinion given in a doubtful case without observing this precaution is of no value. Functional and false murmurs often disappear when the patient lies down, whilst an organic murmur, especially that of mitral stenosis, may be only audible in that position. A murmur which suddenly and completely disappears when the patient lies down, provided there is not at the same time any marked alteration in the pulse-rate, is almost certainly not due to organic disease of a valve.

5. It is important in all cases to auscultate the trachea. A murmur audible in the trachea may be due either (1) to the con-

duction of the murmur of aortic stenosis; or (2) to the impulse of an aneurysm; or (3) to the impulse of the heart causing an air wave in the bronchi and trachea, this latter being by far the most common cause of tracheal murmurs. Mitral murmurs are not audible in the trachea.

6. False murmurs often disappear completely when the breath is held.

7. The state of the pulse may at once negative a suspicion of organic disease founded on the presence of a murmur.

J. K. F.

CHAPTER III

DISEASES OF THE TRACHEA

PAGE

Acute inflammation. Acute tracheitis 71
Chronic tracheitis 72
Diphtheria 73
Tuberculosis 74
Syphilis 76
New growths 76
Stenosis 77
  Symptoms and diagnosis 77
  A. Stenosis from disease originating within the trachea . . 78
    Stenosis from syphilis 78
    „ „ tracheotomy tube 79
  B. Stenosis from compression 80
    Enlargement of thyroid gland 80
    Enlarged glands 81
    Aneurysm and tumour 82
  C. Stenosis from extension of neighbouring disease . . 83
  Treatment of stenosis 83
Perforation of the trachea 83
  Symptoms 85
Foreign bodies in the trachea 85
  Symptoms 86
  Treatment 86

Acute inflammation. Acute tracheitis.—Catarrhal inflammation of the trachea usually accompanies a similar condition in the larynx or bronchi. An ordinary 'cold' may first give evidence of its presence by producing a catarrh of the pharynx: this may extend to the larynx, and subsequently travel down the trachea to the bronchi. But in some cases the inflammation of the trachea is primary, and the change may be localised there and constitute a definite affection.

Symptoms.—A sense of soreness—or, as it is often termed, 'a raw feeling'—along the trachea, and particularly behind the sternum, is the most prominent symptom. This will be accompanied by a harsh dry cough, and possibly by pain on swallowing. If the affection is limited to the trachea, the voice will not be affected. Such general symptoms as ordinarily accompany a severe cold will be present. On laryngoscopic examination it may be possible ve the reddened and swollen condition of the mucous membrane, and in some cases small ulcers have been present. Provided the change does not extend to the bronchi, a favourable termination may usually be looked for in the course of a few days, but occasionally a condition of chronic inflammation supervenes and may require more prolonged treatment.

Treatment.—If the patient is anxious to be quickly rid of his ailment he will be well advised to remain indoors in a warm room (temp. 65° F.) with a bronchitis kettle on the fire. Many, however, regard the affection as too trivial to justify absence from work. A hot linseed-meal poultice applied over the trachea or a mustard-leaf at the root of the neck often gives relief. Steam inhalations with compound tincture of benzoin (ʒj ad ⊙j of boiling water) have a soothing effect upon the inflamed mucous membrane; they should be used for about ten minutes at intervals of an hour throughout the day. A hot mustard bath for the feet and a hot drink at bedtime are domestic remedies of approved value. A Turkish bath acts well in some cases, but we have known the dry heated air prove very irritating to the inflamed trachea. A saline diaphoretic mixture with a sedative (liq. ammon. acet. ʒiv; spir. ætheris nitrosi ♏xx; tinct. camph. co. ♏xx; aq. camph. ad ℥j) may be taken every four hours, and a saline purgative should be administered.

Chronic tracheitis.—This may be a sequel of an acute inflammation which has probably been neglected, or it may accompany chronic bronchitis, particularly in the aged. It should be treated on the lines above laid down.

There is also, however, a very rare form of chronic inflammation of the trachea attended by great thickening of the mucous membrane, which is a much more serious affection. In some of the few cases of this kind which have been recorded the disease has probably been due to syphilis. The following case is an example of chronic tracheitis, and its recital will best describe the symptoms and complications which characterise it, and the treatment which may be necessary.

Edith B., æt. 25, admitted into the Brompton Hospital December 5, 1887, under the care of Dr. Percy Kidd.

Five years ago the hair became thin, but she had no sore throat, rash, or other symptoms of syphilis. Cough has been present for four years. Two years ago the patient first noticed inspiratory stridor. Dyspnœa has of late been increasing, and she now has orthopnœa at night. She has had several attacks of bronchitis during the period covered by the illness.

Expectoration is very slight in amount. There has been no hæmoptysis. Considerable emaciation has occurred, and she has had occasional night sweats. There is no glandular enlargement, and no dysphagia.

Examination of larynx.—The vocal cords were healthy and freely movable. Low down in the trachea, apparently about half an inch above its bifurcation, a yellowish-white swelling was seen on the posterior wall. This was believed to be a new growth, and the treatment adopted was undertaken with a view to its removal.

On December 16 the patient was anæsthetised, and Mr. Godlee performed tracheotomy below the isthmus of the thyroid gland. The third, fourth, fifth, and sixth rings of the trachea were divided, the incision extending to the episternal notch. A prominent fleshy-looking ridge was seen on the posterior wall of the trachea extending downwards almost to the bifurcation. There was no definite isolated tumour. A probe was passed without marked difficulty into each main bronchus. Just above the bifurcation the swelling appeared to be more prominent than elsewhere. The upper part of the trachea was quite healthy. The surface of the prominence was cauterised with a galvano-caustic wire as far as its lower border.

The patient was very seriously ill for five weeks following the operation, which was followed by bronchitis and broncho-pneumonia. Subsequently pericarditis supervened, but ultimately recovery was complete and the stridor disappeared.

On the day of the patient's discharge the bifurcation of the trachea was distinctly visible with the aid of the laryngoscope; there was no bulging there, and the part presented a normal appearance.

Microscopical examination of a small portion of the growth showed that it was covered with stratified epithelium in place of the normal columnar layer.

Diphtheria.—In this disease an exudation commencing in the larynx very often extends to the trachea, and thence to the main bronchi and their branches, even the smaller tubes being affected. In the trachea the membrane may form a complete cylinder loosely attached to the tube, but in the smaller bronchi it is usually soft and puriform.

In the accompanying drawing (fig. 44) the appearances above described are illustrated.

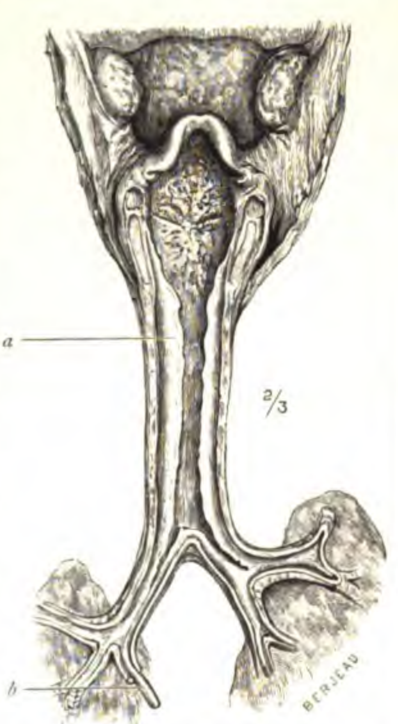


Fig. 44.—Diphtheria of the Larynx, Trachea and Bronchi

a and *b*, membrane in the trachea and bronchi.

Symptoms.—In a case of laryngeal diphtheria it is usually only inference from the increasing severity of the dyspnœa, the onset of cyanosis, and the more marked retraction of the lower ribs and interspaces, that the membrane has extended to the trachea and bronchi.

Treatment.—This condition indicates the necessity for tracheotomy. When the operation has been performed, membrane will be seen within the trachea, and as much of it as possible should be removed with a feather before the tube is inserted. Casts of the lower part of the trachea and larger bronchi may subsequently be expectorated or removed in a similar manner. Such cases are not hopeless, but the prognosis is very grave.

Tuberculosis.—Tubercular disease of the trachea is practically unknown as a primary affection, but is by no means uncommon as a complication of pulmonary tuberculosis. It is usually met with in cases in which the larynx is also involved, but may occur without any lesion in the latter site. Both membranous and cartilaginous portions are usually together affected, but the anterior wall may show the most advanced disease. Infiltration of the mucous and submucous coats of the membranous portion is followed by deep ulceration and thickening. Small rounded superficial erosions or ulcers, limited to the mucous membrane, are often seen over the cartilages. As the disease advances, these unite and lay bare the cartilages. This change, combined with much deeper and more extensive ulceration and swelling of the intervening tissue, often gives the anterior wall the appearance of being formed by successive rings from above downwards, the smooth surface of the bare cartilage alternating with a ring presenting a coarsely granular aspect. In fig. 45 the appearances just described are illustrated, and the alternating smooth and roughened rings are well seen.

The extent of the disease varies. In very advanced cases the whole length of the tube may be involved, and the lesion may extend into the main bronchi. Perforation may occur into the cellular tissue of the neck and be followed by subcutaneous emphysema.

The cases of pulmonary tuberculosis in which lesions of the trachea are of most frequent occurrence are such as have run a rapid course and have been characterised by abundant expectoration of a thin purulent character. Similar secretion can be obtained post mortem, on pressure from the cavities and areas of infiltration and softening within the lungs. The lesions are occasionally confined to the trachea, or are more advanced there than in the larynx.

In 214 consecutive post-mortem examinations[1] on cases of pulmonary tuberculosis the trachea was affected, and the larynx escaped in 13, whilst in 28 of the cases both larynx and trachea were together involved.

Symptoms.—As a rule the symptoms due to the accompanying pulmonary disease are so urgent as to mask those of the tracheal complication.

---

[1] *P. M. Register*, Brompton Hospital, 1893-94

When the larynx is also affected it is still less likely that it will be possible to recognise the tracheal lesion by any definite symptoms. Pain in the tracheal region may be complained of, and the cough in such cases is usually extremely severe.

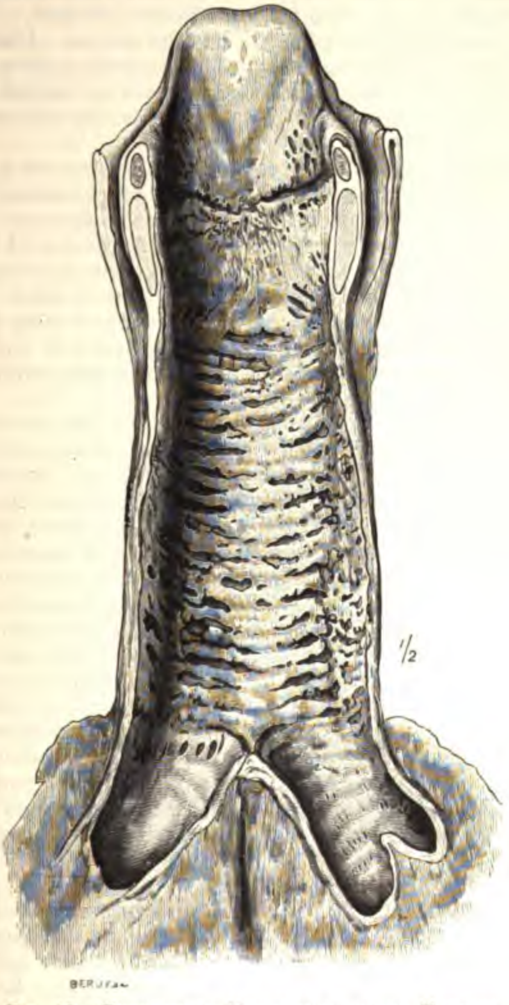


FIG. 45.—TUBERCULAR ULCERATION OF THE TRACHEA

Treatment.—A cocaine spray (2 to 5 per cent.) may temporarily diminish the pain and distressing cough which usually accompany this affection. An insufflation of a powder consisting of morphine gr. $\frac{1}{6}$ with sacch. lactis gr. ij is of great service in advanced tubercular ulceration of the larynx, and may also be used for a similar condition of the trachea. Sedative inhalations of benzoin, hyoscyamus, hop, conium, or chloroform are also of service. The cough may also be relieved by the inhalation of ten minims of a 10 per cent. solution of menthol dropped on to the sponge of an oro-nasal inhaler, which may be worn for some hours at a time. In cases in which the condition of the larynx permits the use of intratracheal injections, it is possible that a solution of menthol (10 per cent.) in olive oil (ʒj) might give relief, but we have no actual experience of this mode of treatment in cases of tubercular ulceration of the trachea.

Syphilis.—The trachea may share in the catarrhal symptoms which occasionally affect the bronchi in the secondary stage of syphilis. Nothing very definite is known as to the exact nature of the lesion in such cases; it is possibly similar to that of the larynx, viz. a formation of mucous plaques. Cough and expectoration are almost invariably present. The trachea is often the site of tertiary syphilitic lesions, and as the majority of cases of stenosis from cicatricial contraction are of this nature, it will be more convenient to discuss the morbid anatomy of the condition under that heading.

Treatment.—The presence of lesions in the trachea in the secondary stage of syphilis may be regarded as a proof either that mercurial treatment in the primary stage has been neglected or has not been sufficiently prolonged, or that the general health of the patient is impaired. In the former case a course of mercurial treatment of from one to two years' duration is essential; in the latter the desirability of such treatment must be left to the judgment of the medical attendant. Iodide of potassium in gradually increasing doses may be combined with the mercury. No effort should be spared to maintain the general health at as high a standard as possible, as under such a condition antisyphilitic treatment is much more likely to be successful. In the tertiary stage of syphilis it is of course hopeless to expect that well-marked stenosis due to the contraction of newly formed fibrous tissue will disappear under treatment by mercury or iodide of potassium, but their use may favourably influence the course of lesions which have not advanced to this stage, and is certainly worthy of prolonged trial.

New growths.—New growths are not often met with in the trachea. Papilloma may occur and may be successfully removed after tracheotomy.[1] A malignant growth may originate in the trachea, or, what is far more common, a primary cancer of the œsophagus may involve the trachea by extension. It will at first cause dyspnœa and stridor, and, after softening and ulceration, cough with expectoration will follow. Such cases often terminate by the occurrence of diffuse septic pneumonia.

---

[1] See *St. Bart.'s Hosp. Reports*, vol. xviii., by Mr. Butlin. Specimen 1656*a*, St. Bart.'s Hosp. Museum.

Polypi are met with in the trachea, but are very rare. The symptoms will be those of tracheal obstruction, and their severity will depend upon the size of the growth. Osseous and cartilaginous growths have also been observed in the trachea, but such cases are exceedingly rare. It may be necessary, both for diagnosis and treatment, to perform tracheotomy.

Stenosis.—Narrowing of the trachea may be due (*a*) to lesions originating within, (*b*) to pressure from without, or (*c*) to the trachea becoming involved by the extension of morbid processes from neighbouring parts.

Symptoms and diagnosis.—Dyspnœa is the most important symptom of narrowing, to whatever cause that may be due. It comes on gradually, and increases in proportion to the degree of stenosis. When this is considerable the dyspnœa may become paroxysmal in character and be so severe as to threaten life.

Orthopnœa may be present, and short of this the breathing is often more embarrassed when the patient is lying down. Cough may be due to irritation or to the collection of mucus behind the obstruction. The latter condition gives rise to coarse tracheal râles, and when the quantity of mucus is considerable may be the immediate cause of the paroxysmal attacks of dyspnœa. The breathing is accompanied by stridor, chiefly on inspiration, and suggesting laryngeal obstruction; but the larynx on examination may prove to be unaffected, and the stricture of the trachea may be seen. On auscultation over the trachea it may be possible to determine the site of maximum intensity of the sound, and this may be a guide to the position of the stricture; but in some cases the stridor is most marked over the larynx. Apart from the evidence obtained on laryngoscopic examination, the signs indicating tracheal as distinguished from laryngeal obstruction are that in the former condition the 'respiratory excursion' of the larynx will not be increased and the voice, although weak, will not necessarily be otherwise affected.

The position assumed by the patient in the two affections also differs. In laryngeal obstruction the patient as a rule sits up with the head thrown backwards, whereas in tracheal stenosis the head is held forwards with the face looking downwards. The breath sounds, though weak, will be equal on the two sides if the narrowing is confined to the trachea; but it must be remembered that syphilitic lesions often affect one of the main bronchi to a greater degree than the other as well as the trachea.

Aufrecht has recently described a 'mewing' inspiratory murmur, most marked when the patient makes any bodily exertion, as characteristic of tracheal stenosis. The same author has observed that in some cases of tracheal stenosis the harsh bronchial sounds normally audible over the lower end of the trachea are replaced by soft low breath sounds of short duration or are not audible at all.

A. Stenosis from Disease originating within the Trachea

Syphilitic Stenosis.—*Morbid anatomy.*—The disease commences as a localised gummatous infiltration of the mucosa, causing, in the early stage, considerable swelling. The gumma may be absorbed and a cicatrix result, or it may break down and an ulcer form, and this may heal with the production of a scar. Portions of the cartilages may undergo necrosis after perichondritis and may be expectorated, or one or more cartilages may be absorbed. On laying open the trachea by section through the posterior wall it may be found to present one or two or numerous cicatrices, and in severe cases the tube almost from end to end may be covered by white fibrous scars having a radiating appearance. The submucous layer may be extremely thickened, measuring perhaps a quarter of an inch or more on section, and the lumen may be much diminished. The narrowing may be limited to or most marked at one spot, or may extend for some distance. The tube may be dilated above and below the seat of the stricture. The disease is

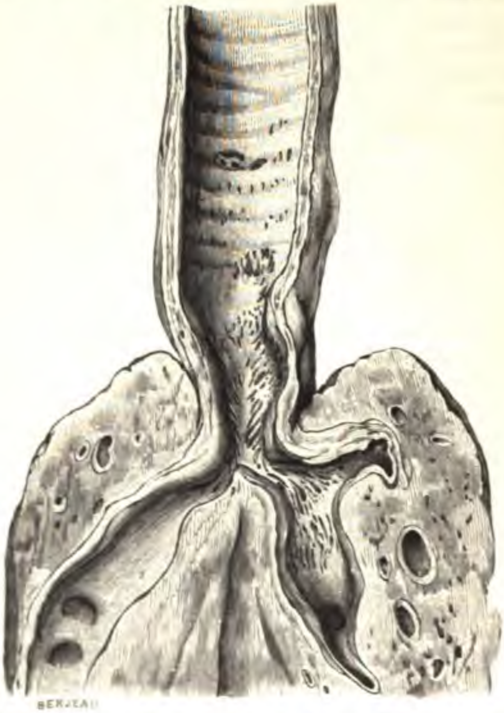


Fig. 46.—Syphilitic Stenosis of the Trachea and main Bronchi

most often situated at the lower end of the trachea. The accompanying drawing (fig. 46) of a specimen in the museum of Brompton Hospital illustrates these changes.

The patient was a woman aged 25, whose case is reported in full in the chapter on Pulmonary Syphilis (see p. 447). The lesions in the trachea are thus described:[1] 'A few small scars in the subglottic portion of the larynx. The lower half of the trachea was marked by numerous stellate puckered cicatrices, involving both membranous and cartilaginous portions, but especially the latter. The origin of the left bronchus was represented by a small opening, just admitting a probe. The surrounding parts of the tracheal wall were extremely fibrous and puckered. There was scarring in the right bronchus, about the origin of the upper lobar branch.'

The following is a description of the condition present in a case of syphilitic stenosis of the trachea of many years' duration, illustrated in fig. 47.

'On the anterior surface, about an inch from the larynx, is a patch of superficial ulceration. About two inches below this point the trachea is markedly constricted by a well-defined narrow ring of cicatricial tissue with a sharp margin. Immediately above the bifurcation is another constriction, produced by extensive scarring and puckering of the wall; between the two the lumen is narrower than normal. At the level of the lower stricture there is a mass of indurated and pigmented glands firmly uniting the trachea to the aorta. There is no ulceration of the lining membrane, or other evidence of recent change.'

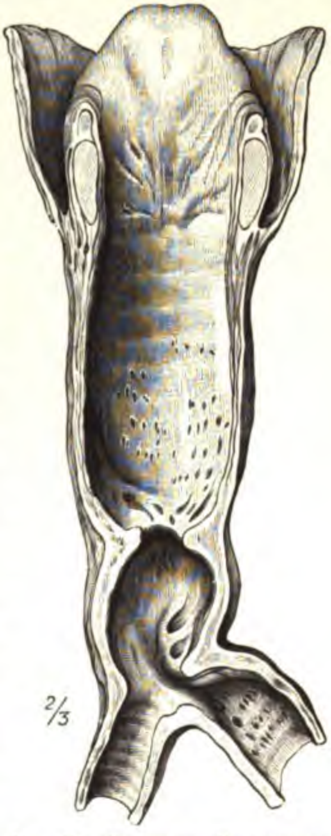


FIG. 47.—SYPHILITIC STENOSIS OF THE TRACHEA

STENOSIS FOLLOWING TRACHEOTOMY.—The irritation caused by the long-continued presence of a tracheotomy tube may lead to a growth of granulation tissue which may become organised into fibroid tissue and undergo contraction, the result being a narrowing of the trachea after the cicatrisation of the wound. The term 'posttracheotomic vegetations, or polypi,' is applied to these growths. The condition is most frequently met with in male children, from a fortnight to a month after the wound has healed.

---

[1] Reported by Dr. Percy Kidd, *Path. Soc. Trans.* vol. xxxvii. p. 111.

B. Stenosis from Compression

Enlargement of the thyroid gland, either as a whole or in part, may cause narrowing, varying in kind and degree.

Fig. 48 illustrates the alteration in the lumen of the tube which may result from enlargement of the isthmus and both lateral lobes of the thyroid. The transverse diameter is seen to be diminished to such a degree that the walls are almost in contact. Below the site of narrowing the trachea is dilated. The middle lobe was spherical, as large as a tennis ball, and in an advanced stage of colloid degeneration. Both lateral lobes were considerably enlarged, and were compressed by the cervical fascia and muscles. The lower part of the trachea was coated with a false membrane, consisting of inspissated pus. The goître had been present for

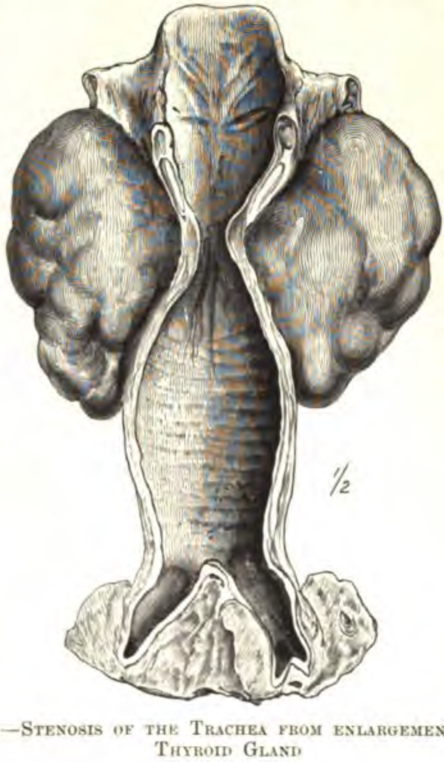


Fig. 48.—Stenosis of the Trachea from enlargement of the Thyroid Gland

eight years. Death was due to extensive tubercular disease of the lungs.

The alteration in the axis and lumen of the trachea illustrated by figs. 49 and 50[1] was produced by a growth containing within a fibrous capsule glandular structure similar to that of the thyroid (? parathyroid). The specimen was taken from a man aged 36, who was found dead in a street adjoining the Middlesex Hospital. No history of the case could be obtained. The tumour was possibly a parathyroid embedded in a thick fibrous capsule. The inner segment was firmly fixed to the trachea from the fourth to the ninth ring. The central part of the tumour exhibited under the microscope the characteristic structure of the thyroid gland. The capsule consisted of dense laminæ of fibrous tissue. The cyst on the thyro-hyoid membrane (fig. 49) was due to an enlargement of the bursa which is usually present between the body of the hyoid bone and the thyro-hyoid membrane.

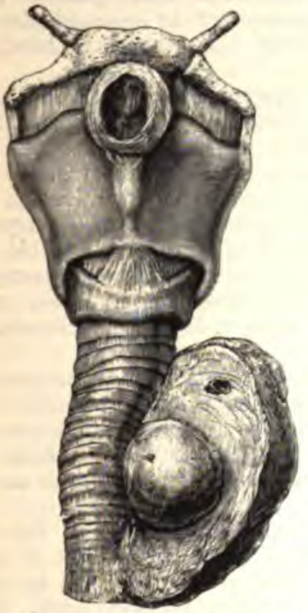


FIG. 49.—A GROWTH CONTAINING THYROID GLAND STRUCTURE COMPRESSING THE TRACHEA

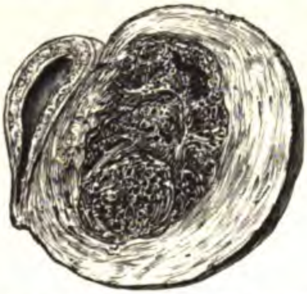


FIG. 50.—SECTION OF THE GROWTH AND OF THE CONTAINED TUMOUR AND TRACHEA, SHOWING THE DEGREE OF STENOSIS (NATURAL SIZE)

The effects produced by an enlargement of one lateral lobe of the thyroid are similar to those illustrated by figs. 49 and 50.

The trachea may be compressed from the front by an enlargement of the isthmus of the thyroid gland, and in cases in which the enlargement is extreme its lumen may be almost obliterated.

ENLARGED GLANDS may compress the trachea and cause thinning, erosion or narrowing. In the neck the enlargement may be due to various causes. It is rare in adults for tubercular glands to cause serious compression, although in children this may occur. Mr. Makins has reported a case[1] (a child æt. 3 years) in which a suppurating gland caused death by pressure on the trachea without the occurrence of perforation. In a case in an adult recently under the writer's care in the Brompton Hospital, glands enlarged from tuberculosis caused such a degree of displacement of the larynx and trachea that the apex of the thyroid cartilage lay three-fifths of an inch to the opposite side of the median line. In *syphilis* the glands of the neck may enlarge and compress the trachea, and the enlargement may even be so considerable as to suggest a diagnosis of lymphadenoma. The lower part of the trachea within the thorax may be compressed by *enlarged glands* or by a *mediastinal growth*, and in both cases the main bronchi may also be involved.

---

[1] See *Brit. Med. Jour.* March 2, 1895, p. 463, Clinical Lecture by Mr. J. Bland Sutton.

An ANEURYSM of the carotid or innominate artery, or of the transverse part of the arch of the aorta, may compress the tube, and an abscess due to caries of the spine may have a similar effect.

Symptoms and diagnosis of compression.—In the majority of cases the cause is obvious, but, if not, a careful examination of the upper part of the thorax must be made for signs of aneurysm or tumour. The presence of 'tracheal tugging,' with feeble or absent breath sounds over the left upper lobe, points very strongly indeed to aneurysm. The differential diagnosis of these two conditions is fully discussed in the chapter on Mediastinal Tumours (p. 691).

It is, however, possible in cases of compression by an aneurysm or growth that narrowing of the trachea may be accompanied by paralysis of the intrinsic muscles of the larynx from implication of the vagus or of one or both recurrent laryngeal nerves, or by fixation of one vocal cord from local disease of the larynx.[2] In such cases the diagnosis may present some difficulty.

The patient from whom the specimen illustrated in fig. 47 (p. 79) was obtained was long believed to be the subject of aneurysm of the aorta, owing to the fact that in addition to the tracheal stridor there was fixation of the one vocal cord. On autopsy this was found to be due to local tuberculous disease, and not, as was expected, to pressure on the recurrent laryngeal nerve.

It may also be difficult to distinguish between the symptoms arising from the presence of a malignant growth originating in or invading the trachea, and narrowing due to syphilis. The longer the tracheal symptoms have lasted without producing definite signs of stenosis the more probable it is that they are due to syphilis.

Marked stenosis, when the symptoms are of recent date, points to the presence of a growth.

---

[1] *Path. Soc. Trans.* vol. xxxv.

[2] *Ibid.* vol. xxiv. p. 42.

C. STENOSIS FROM EXTENSION TO THE TRACHEA OF DISEASE IN NEIGHBOURING STRUCTURES

This occurs most frequently in malignant disease of the œsophagus, and owing to the fact that in some cases of that nature dysphagia is very slight or even absent, the origin of the disease in the œsophagus often remains unsuspected up to the time of the patient's death. Primary malignant disease of the bronchial or mediastinal glands may also invade the trachea and cause narrowing of its lumen.

Treatment of stenosis.—It may become necessary to resort to tracheotomy in the event of the narrowing tending towards occlusion.

It is scarcely necessary to point out that before the operation is decided on it is absolutely necessary to determine the site of the obstruction, and to make sure that the trachea can be opened below it. A careful examination of specimens of tracheal syphilis will show that in such cases the operation can rarely be of much service, owing to the fact that the disease is either not sufficiently localised or is situated too low down. It may, if unsuccessful, do harm, as it renders the cough less effective; and as in such cases expectoration is often profuse, if it is allowed to accumulate, inflammation and gangrene of the lungs is very likely to follow.

In cases of compression of the trachea associated with bilateral paralysis or spasm of the intrinsic muscles of the larynx from the pressure of an aneurysm or tumour upon one vagus, or from the implication of both recurrent laryngeal nerves, tracheotomy may give great relief for a time.

Paroxysmal dyspnœa from compression of the trachea by an aneurysm may be relieved by inhalations of oxygen. In a case recently under the care of the writer this treatment alone gave relief. Subcutaneous injections of ether (♏ x) or strychnia (gr. $\frac{1}{30}$–$\frac{1}{50}$) or inhalations of chloroform may also be tried. A mixture containing ether and ammonia may be given as a stimulant, and will also aid the patient in getting rid of accumulated mucus. In cases in which this latter symptom is not prominent, and where there is considerable nervous distress, morphia may be injected subcutaneously. If, as sometimes happens, the true cause of the paroxysms of dyspnœa has not been recognised, and a patient suffering from compression of the trachea by an aneurysm of the transverse arch of the aorta has been treated for 'bronchitis and emphysema,' rest in bed with gradually increasing doses of iodide of potassium will be quickly followed by great relief to the symptoms.

PERFORATION OF THE TRACHEA

hea may be perforated by a caseous lymphatic gland or sm, or by a growth originating in the œsophagus. A us may also penetrate the trachea, but such an event rrence.

If a gland, after perforating the tube, should undergo gradual disintegration, a diffuse broncho-pneumonia or tuberculosis may be set up, and may prove fatal. If, as occurred in a case observed by the writer, a communication is simultaneously established with the œsophagus, diffuse gangrene of the lung may follow.

In rare cases the greater part of the gland is discharged as a solid mass into the trachea, an event which may be immediately followed by death from asphyxia. Fig. 51 illustrates such a case. The patient was a boy æt. 7 years, admitted into the Brompton Hospital for a croupy cough and slightly stridulous breathing. He was apparently in fair health in the afternoon and was walking about. During the night he suddenly jumped up in bed, screamed, and gave a croupy cough. Death from asphyxia occurred in less than ten minutes.[1]

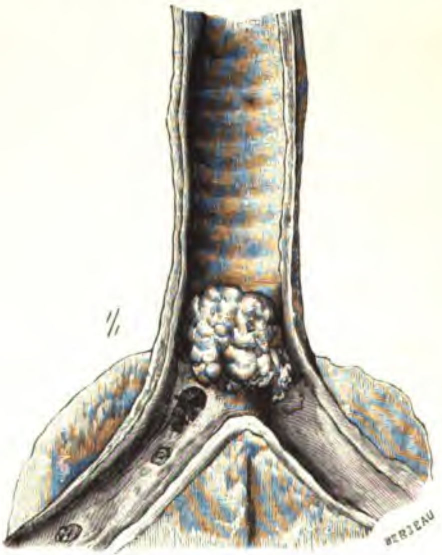


FIG. 51.—PERFORATION OF THE TRACHEA BY AN ENLARGED GLAND, AND DISCHARGE OF THE GLAND INTO THE TRACHEA

A calcareous gland may rupture into the trachea and also into the aorta, and sudden death may occur from hæmorrhage into the lungs.

The accompanying drawing (fig. 52) illustrates the effect produced upon the trachea by perforation of an aneurysm of the innominate artery. The small nipple-like projections indicate points where rupture was impending from gradual erosion of the wall.

---

[1] The case is reported by Dr. Percy Kidd in the *Pathological Society's Transactions*, vol. xxxvi. p. 104.

The specimen was taken from a man who was brought into the Middlesex Hospital dead. No history of the case could be obtained.

Symptoms.—The rupture of an aneurysm will be immediately followed by hæmoptysis, which is usually quickly fatal; but in some cases only slight leakage occurs at first and life may be prolonged for a few days or more. The hæmorrhage may cease and recur. The results of perforation by a gland have already been described.

Perforation by an abscess will be followed by the expectoration of the pus, but probably a certain quantity will be inhaled, and if death does not immediately occur pneumonia may supervene.

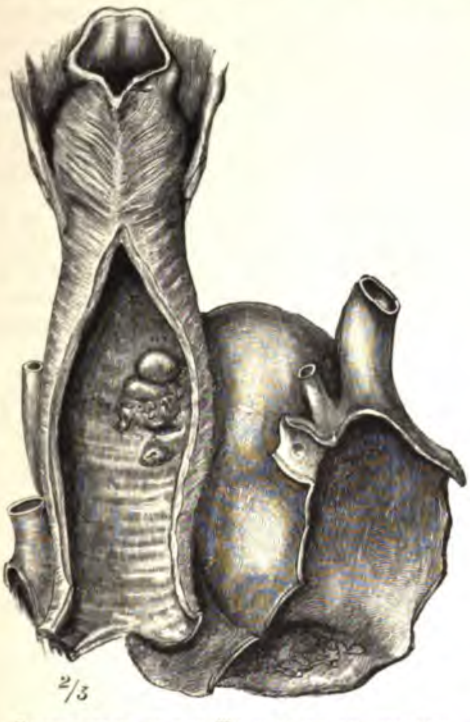


FIG. 52.—PERFORATION OF THE TRACHEA BY AN ANEURYSM OF THE INNOMINATE ARTERY

FOREIGN BODIES.—A foreign body is more likely to lodge in one of bronchi, especially the right, than in the trachea, as after passing larynx it is not usually arrested above the bifurcation. A great iety of substances too numerous to mention have, however, been nd in the trachea.

A coin may become impacted in the trachea, and it may be possible, aid of the Röntgen rays, to determine its exact position.

a case under the care of the writer a portion of a tumour—a sarcoma—which had been moulded by extension into a large bronchus into the form of a solid cylinder, became detached, and was driven during an attack of coughing into the trachea, and becoming impacted below the vocal cords, caused sudden death from asphyxia.

Symptoms.—Museums contain a large number of specimens showing portions of meat and other substance impacted in the larynx and extending through the glottis into the trachea. In such cases death from asphyxia is immediate. When the body is of smaller size and passes the larynx its presence in the trachea causes dyspnœa, varying in degree with the amount of obstruction to the breathing. Violent spasm is excited, particularly by bodies with roughened surfaces, and this by still further narrowing the tube increases the dyspnœa. In all the cases here described sudden asphyxia was produced by the body being forced by violent cough against the under surface of the glottis. A body such as a fish bone may become fixed in the wall of the trachea and may not cause obstruction, although it will give rise to pain, especially when the trachea is moved in the act of swallowing.

Treatment.—If the foreign body can be seen on tracheoscopic examination, or if its position has been determined by the aid of the Röntgen rays, an effort may be made to remove it by the use of long tracheal forceps, an operation requiring for success considerable skill and some good fortune. This failing, tracheotomy should be performed.

J. K. F.

CHAPTER IV

BRONCHITIS

	PAGE
Classification	87
(*a*) ACUTE BRONCHITIS OF THE LARGER TUBES	87
Morbid anatomy	87
Etiology	89
Symptoms and course	90
Physical examination	92
Diagnosis	92
Prognosis	93
(*b*) ACUTE BRONCHITIS OF THE SMALLER TUBES. Capillary bronchitis	94
Morbid anatomy	94
Etiology	94
Symptoms	94
Physical signs	95
Course	95
Prognosis	95
Diagnosis	96
Treatment of acute bronchitis of the larger tubes	97
" *of capillary bronchitis*	99

A catarrhal inflammation of the bronchial tubes, of which the chief symptoms are cough, expectoration, and dyspnœa.

Classification.—The varieties of bronchitis are so numerous that a single description will not suffice for all.

The course of the disease is the basis for the first subdivision into the *acute* and *chronic* forms. The size of the tubes affected has such an important influence upon the severity of the attack that a separate account is necessary of *Bronchitis of the Smaller Tubes.* The disease occurs as a complication of so many and such diverse disorders as to require consideration under the head of *Secondary Bronchitis*; and, lastly, a special and rare form of inflammation of a different character—*Plastic Bronchitis*—is so clearly marked off from any of the preceding varieties as to constitute practically a separate disease. The disease will be described under the following headings: 1. Acute Bronchitis (*a*) of the Larger Tubes; (*b*) of the Smaller Tubes. 2. Chronic Bronchitis. 3. Secondary Bronchitis. 4. Plastic Bronchitis.

(*a*) ACUTE BRONCHITIS OF THE LARGER TUBES

Morbid anatomy.—The first change observed in inflammation of the bronchi is hyperæmia of the vessels of the inner fibrous coat, which becomes swollen, relaxed, softened, and infiltrated with lymph cells. This is followed by œdema of the basement membrane, the surface of which now presents a wavy and folded appearance.

These changes in the basement membrane loosen the attachment of the ciliated columnar epithelium, which separates in patches, and is shed into the lumen of the tube, whence it is either expectorated or inhaled into finer tubes. From the flat cells of the deeper layer of the epithelium thus exposed, new cells presenting various transitional forms, oval or pyriform, continue to be produced, cast off, and expectorated, along with the masses of leucocytes which have left the congested vessels. As the change progresses, all the structures of the bronchial wall become involved, and leucocytes infiltrate the muscular and outer fibrous coats, and in the latter many larger flattened cells are seen: these probably arise from proliferation of the lymphatic endothelium (Hamilton).

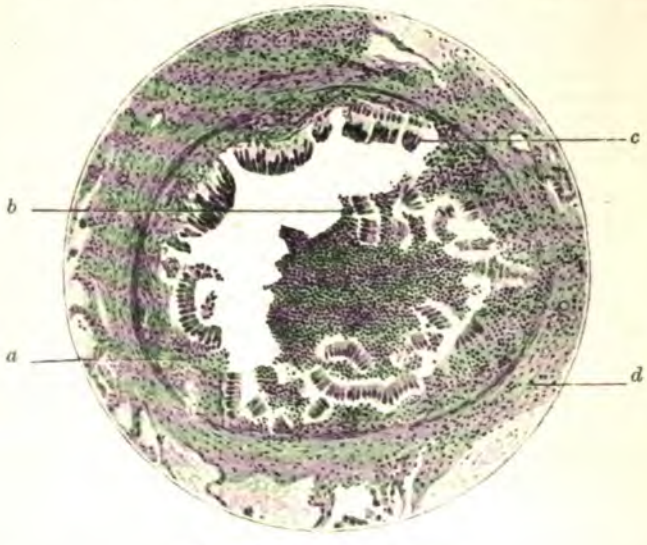


FIG. 53.—ACUTE BRONCHITIS

Transverse section of medium-sized bronchus, showing (*a*) submucous infiltration with partial loss of epithelium; (*b*) detached epithelium in lumen of bronchus embedded in cellular exudation; (*c*) epithelium intact; (*d*) peribronchial infiltration.

The hyperæmia of the mucous glands of the bronchi leads to an enormous increase in the secretion and discharge of mucus, the epithelial and secreting elements of the glands undergoing at the same time a process of desquamation.

These changes naturally cause considerable narrowing of the tubes, some of the finer bronchi being completely occluded. Around the latter, areas of collapse and broncho-pneumonia are frequently found, especially in children, the occluded tubes being not uncommonly dilated. The lymphatic glands along the main bronchi and at the root of the lung are enlarged from hyperæmia, and the nerve ganglia are congested.

On post-mortem examination the lungs are usually large and heavy, but nowhere completely consolidated. Areas of collapse, if present, are sharply defined and of a deep purple colour. The bronchial mucous membrane presents a velvety appearance and still shows the effect of hyperæmia, but this is probably in all cases less marked than during life. The larger tubes contain much mucopurulent secretion, and on section beads of pus may be squeezed from the finer bronchi. The posterior part of both the upper and lower lobes may be almost solid from œdema, collapse, and congestion, a more or less frothy watery fluid pouring out on pressure. Emphysema is commonly present upon the surface and along the margins, varying in extent according to the nature of the case, being often in part a relic of previous attacks of bronchitis.

The right cavities of the heart are dilated and filled with blood, and the tricuspid valve may show marked incompetence. When the patient has succumbed to an acute attack which has supervened upon a chronic condition, an event of by no means uncommon occurrence, hypertrophy of the right ventricle will also be present. Dilatation of the large vessels carrying blood to the right side of the heart, and congestion of the organs, which they drain either directly or indirectly, are necessary consequences of the cardiac lesions.

Etiology.—A *chill* from exposure to cold is the immediate exciting cause in many cases of bronchitis. Its mode of action in this as in other affections is obscure, and its importance as a factor in the causation of disease has possibly been over-estimated; but as regards catarrh of the respiratory passages the belief in it remains unshaken. The effects of a chill are diverse. This may be explained by the fact that almost every individual has his 'weak point,' and in some at least this is an undue susceptibility of the mucous membranes to catarrhal inflammation. In cold and damp climates, and especially in the winter months and during the prevalence of cold winds, which in this country blow most commonly from the east and north-east, attacks of bronchitis are of most frequent occurrence.

Impurity of the air, from whatever cause arising—*e.g.* overcrowding, deficient ventilation in gas-lit workrooms, the presence of fog, or of irritating dust in suspension in the air—is another important factor in producing the disease, the condition acting either by its direct effect upon the mucous membrane, or by lowering the resisting power of the individual exposed to the injurious influence. That previous attacks increase the liability to the affec- n is undoubted, and that this is in great part due to the emphysema ehind is highly probable.

ne special liability of the subjects of *spinal curvature* to bronchitis arises from the distortion of the chest thereby entailed, and its effect upon the lungs. In cases of lateral curvature the posterior part of the lung on the convex aspect of the curve tends to become congested and collapsed from narrowing of the chest, and the opposite lung undergoes compensatory enlargement. Cough is excited by the congestion, emphysema follows, and the liability to bronchitis thus becomes established.

Obstruction to the circulation of the blood through the lungs, such as occurs in chronic valvular disease, by inducing pulmonary congestion, becomes an important etiological factor in bronchitis.

Weakening and destruction of the elastic tissue of the lungs, and the change which follows thereupon—i.e. *emphysema*—by giving permanence to conditions favourable to the disease, also increase the tendency to its recurrence.

Whatever views may be held as to *heredity*, it will hardly be denied that parents may transmit to their offspring a constitution lacking in the resisting power to disease possessed by the healthy organism. It is at any rate probable that this lack of resisting power may specially affect certain organs or tissues, such as those of the respiratory system, or the mucous membranes. If this is so, it follows that a peculiar liability to catarrhal inflammation may be inherited, a conclusion fully justified by clinical experience.

The presence in the body of some *toxic materials*, whether derived from without, as in the case of many of the acute specific diseases, especially measles and typhoid fever, or from within, as in gout and chronic Bright's disease, is also a frequent cause of bronchitis.

Symptoms and course.—An attack of acute bronchitis may vary in severity from that of a mild catarrhal inflammation of the trachea and larger bronchi from which recovery follows in a few days, to that of a severe or fatal illness; but the cardinal symptoms—cough, expectoration, and dyspnœa—are never wholly absent. This statement may pass, although it is true that children below the age of five years do not expectorate, and that the dyspnœa caused by a slight catarrh of the larger tubes may be inappreciable. The *mode of onset* of the attack usually gives some indication of its probable severity. As a rule, when the disease is of a mild character and limited to the trachea and larger tubes, it commences as an ordinary catarrh, either nasal or laryngeal, accompanied by hoarseness and a tickling sensation in the throat, or with a sense of rawness behind the sternum from implication of the trachea. In an individual rendered susceptible by the presence of emphysema, the result of previous attacks, the onset is likely to be attended by a sense of tightness in the chest, an indication that the medium-sized bronchi are primarily affected. In similar cases the disease may present urgent symptoms at a very early stage, beginning with a sense of oppression of the breathing, which quickly develops into marked dyspnœa. In children and old people an attack is often sudden in onset and severe in type, and the liability to a fatal termination is far greater than in early adult and middle life.

The symptoms which usually accompany a chill, such as general malaise and pains, are present; but rigors are of rare occurrence. The degree of pyrexia is moderate, a temperature above 100·5° or 101° F. being unusual in an adult, but by no means so in a child.

Cough is an early symptom. It is at first short, dry, and hacking, and often occurs in severe paroxysms. The cough retains this character until the hyperæmia of the bronchial mucous membrane has given rise to secretion. The *expectoration* at this period consists of scanty, viscid, tenacious mucus, adhering to the surface of the vessel which receives it, and expelled with much difficulty. As it increases in quantity its characters alter, it is got rid of more easily, and the cough is said to have become 'loose.' It is now less viscid and tenacious, frothy from admixture of air, resembles the white of egg, and shows whitish streaks, and may contain streaks of blood. As the disease progresses, the denuded mucous membrane pours out increasing quantities of leucocytes, which give a purulent character to the expectoration. This is now opaque, yellow or yellowish green in colour, tends to form masses, and is expelled with less difficulty but in greater quantity, six to eight ounces or more in the twenty-four hours being by no means an unusual amount. Cough and expectoration are almost always worse after sleep, from the irritation due to accumulated secretion. The character of the sputa should be closely watched, as constituting the surest guide to the changes taking place within the chest.

If, for example, the expectoration, previously free, copious, and mucopurulent, should diminish in quantity, again become viscid, mucoid, and difficult to expel, the patient will almost certainly complain of greater difficulty in breathing, and will probably say that he has 'caught a fresh chill.'

On *microscopical examination* the sputum is found in the early stage to consist of clear fluid, cylindrical and ciliated epithelium, and mucous corpuscles; in the later stage, of large numbers of pus cells, various forms of epithelial cells and large hyaline cells, granular matter, and small oil globules. Pigment granules and blood corpuscles may also be present.

The degree of *dyspnœa* present in a primary attack of acute bronchitis is a measure of the extent of the obstruction of the tubes; but when emphysema, congestion, or œdema of the lungs, the result either of previous attacks or of cardiac failure, co-exist, a moderate degree of obstruction may produce marked difficulty in breathing. In children the respirations often number forty to sixty or more in the minute; but in adults, when the large and medium-sized tubes only are affected, there may not be much dyspnœa when the patient lies quietly in bed, with the shoulders well raised. After a paroxysm of cough, an attempt at exertion, or prolonged talking, the respirations are always quickened.

The symptoms of an attack of acute bronchitis other than those dy mentioned do not need a detailed description; they are of the febrile state—a quickened pulse, headache, thirst, a coated tongue, loss of appetite, usually constipation, scanty high-coloured urine with a deposit of lithates. The skin is at first dry, but quickly becomes moist, and before long there may be free perspiration. This is not, however, necessarily accompanied by any marked relief to the symptoms, as it may be, according to its degree, an indication of failure of vital power.

Physical examination.—On inspiration elevation is seen to be well marked, but simultaneous expansion of the lateral aspect of the thorax is often deficient, and in children recession of the ribs and interspaces in this region may occur from the combined effects of bronchial obstruction and weakness of the cartilages. This is especially common in the subjects of rickets, and is then associated with projection of the lower part of the sternum and recession of the supraclavicular regions. Emphysema of the 'large-lunged' variety, the result of previous attacks, will, if present, be accompanied by its usual signs, of which the barrel-shaped outline of the chest, epigastric pulsation from downward displacement of the heart and over-distension of the right ventricle, are the most evident. On *palpation* rhonchal fremitus may be felt when adherent mucus is obstructing the larger tubes. The *percussion* note may be hyper-resonant from emphysema, whilst dulness at the bases of the lungs suggests the presence of congestion, collapse, and œdema.

Auscultation.—Harsh breath sounds, with prolonged expiration, constitute the typical condition. Absence of the breath sounds over a definite area points to complete bronchial obstruction or to collapse of the lung. Sibilant and sonorous rhonchi are the characteristic adventitious sounds of bronchitis. The former, due to narrowing of the smaller tubes, are uninfluenced by cough; the latter, dependent upon the presence of mucus in the larger bronchi, often disappear or alter their site after cough. They may be present both on inspiration and expiration, but are usually most marked during the latter period. Bubbling râles, fine, medium, or large, which are more common at the bases than elsewhere, indicate the presence of mucus in a more or less liquid condition in the medium-sized and smaller tubes.

By daily examination a careful watch should be kept over the posterior aspect and the bases of the lungs in every case of bronchitis, especially in children and the aged, as post-mortem experience proves that there the changes are usually most marked. This is the effect of the recumbent position, also of the gravitation of fluid, and of the frequent presence of emphysema not only at the margins but also on the posterior aspect of the lower lobes. It must not be at once assumed that medium-sized and fine râles audible at the bases are due to the attack then present, as they may, in the subjects of chronic bronchitis with an acute exacerbation, be relics of previous attacks. Fine crackling râles, either at the bases or elsewhere, usually indicate the presence of œdema, hypostatic pneumonia, or broncho-pneumonia.

Diagnosis.—The diagnosis of acute bronchitis in an adult is rarely attended with much difficulty. Twice, however, in the experience of the writer the same patient has been admitted into hospital suffering from what was supposed to be bronchitis, when the real affection proved to be an acute congestion of the lungs from cardiac failure secondary to *mitral stenosis*. As pointed out by Sir William Broadbent, the presystolic murmur under such circumstances may disappear, and in any case its detection may be difficult owing to the adventitious sounds in the bronchi. The very rapid improvement which usually results from complete rest, the administration of digitalis, and from venesection, and the subsequent reappearance of the murmur lead, as a rule, to a correct diagnosis in a few days. In children the *onset of acute diseases*, such as whooping cough and measles, may be masked for a time by the bronchial inflammation. A convulsive cough leading to vomiting should in all cases excite suspicion of the former, and in the latter the appearance of the rash soon determines the nature of the case.

Miliary tuberculosis of the lungs may usually be distinguished by the higher range of temperature, more persistent pyrexia, more marked prostration, and the tendency to delirium. Adventitious sounds may be entirely absent in tuberculosis, but if present they are not generally at first dry and at a later stage moist as in bronchitis, but bubbling and fine crackling râles may predominate from the outset. Signs limited to one upper lobe, or to the apices of both lungs, even though catarrhal in character, should always suggest the presence of pulmonary tuberculosis rather than of bronchitis. In a child acute bronchitis is less likely than acute broncho-pneumonia to be mistaken for miliary tuberculosis of the lungs.

Prognosis.—As the physician's experience of acute bronchitis increases, he is less ready to assure a sufferer, even in apparently mild cases, that he will be 'all right in a few days.' The duration of the disease is apt to be very much longer than appears at first likely; a mild case may last a fortnight, and, if at all severe, recovery rarely occurs in less than three weeks or a month. When emphysema is present, recovery is almost always slow, the patient being confined to bed for weeks or months, and possibly to the house for the greater part of the winter.

The only cases in which a fatal termination is to be feared are those occurring in young children and old people, or as a complication of chronic bronchitis and emphysema, associated or not with mitral or tricuspid disease. A primary attack of acute bronchitis limited to the larger and medium-sized tubes is hardly ever fatal in a previously healthy subject.

The necessity for a careful examination of the sputum, and the indications to be obtained therefrom as to the course of the disease, have already been stated. The danger signals of acute bronchitis evidence of over-distension of the right side of the heart—cyanosis, distension and pulsation of the veins of the neck—ent dyspnœa or orthopnœa, and a tendency to sink down into the bed, accompanied by cessation of expectoration. This latter symptom is of especially unfavourable import, but we have observed recovery take place after it has occurred. Great frequency with irregularity and intermission of the pulse are also very unfavourable signs. When the extremities become cold, and the skin is covered with profuse clammy perspiration, or the mind wanders, and coma threatens, it is obvious that recovery is almost hopeless. Even when improvement occurs it is well to remember that relapses are of common occurrence, and to be cautious in prognosis. The patient should not be pronounced to be well until the cough and the adventitious sounds have disappeared.

The **treatment** of the various forms of bronchitis is discussed on pages 97–102 and pages 108 and 117.

(*b*) ACUTE BRONCHITIS OF THE SMALLER TUBES

(*syn.* CAPILLARY BRONCHITIS, SUFFOCATIVE CATARRH)

This is an affection of a very different degree of severity from that just described. It is never a slight disorder, or otherwise than a dangerous and possibly fatal malady.

Morbid anatomy.—The changes which occur are similar to those described in the previous chapter, the urgency of the symptoms depending upon the great obstruction to respiration caused by inflammation of a large number of the smaller tubes.

The lungs are either over-distended with air, or extensive emphysema may be present, and this may be, in great part, of recent origin. Areas of collapse of varying size may be seen, involving, in some cases, the greater part of a lobe. On section fine beads of pus may be expressed from the smaller tubes, some of which are often dilated. Others are found to be completely blocked by secretion, which may assume the form of a more or less organised exudation, and the lining membrane is invariably markedly injected. The presence of small, rounded, faintly yellow areas indicates that the alveoli have shared in the inflammatory process (bronchopneumonia). The changes present in the medium-sized and larger tubes and in the heart and other viscera are similar to those described in the preceding section, but the latter are usually more marked owing to greater degree of venous obstruction.

Etiology.—This variety of the disease does not differ as to causation from the milder type, and its occurrence probably depends upon the defective resisting power of the individual attacked. It is especially common in infants and young children and in the aged. In mid-adult life it occurs, in our experience, most often in fat subjects who have had previous attacks of bronchitis, and especially in women. The very small capacity of the chest in fat women is often remarkable. Those addicted to alcoholic excesses are certainly more liable to this severe form of the disease than the temperate.

Symptoms.—The affection of the smaller tubes may be primary, or the disease may extend downwards from the larger bronchi; the latter is usually stated to be the most common mode of onset, but in our experience the contrary has been the case. The onset is often sudden, a rigor may occur, or in a child a convulsion may take place. The cough is very frequent, and may be paroxysmal in character and accompanied by pain in the chest. It is at first dry and may remain so, expectoration being throughout absent, or it may be small in quantity and expelled with great difficulty. The alæ nasi are widely dilated and the dyspnœa is severe, but on examination of the chest there may be few signs to account for it. Orthopnœa occurs early, the respiratory efforts and abdominal movements being markedly exaggerated. The face quickly becomes cyanotic, but the underlying pallor points to early cardiac failure, and there is marked prostration. Anxiety and restlessness are present almost from the outset, and often give place to delirium towards the close. The skin of the body may be bathed in sweat, whilst the lower extremities are cold. The pulse is rapid—120 or more. The temperature is much higher than in the ordinary form of the disease, even 104° F. being by no means uncommonly observed in children.

Physical signs.—The upper part of the chest is distended, whilst at the same time the recession of the lower intercostal spaces and of the epigastric, hypochondriac, and supraclavicular regions, shows how great is the obstruction to the entrance of air into the alveoli. The percussion note is either unaltered or hyper-resonant from emphysema, or there may be dulness at the bases from pulmonary collapse, a condition especially likely to occur in children. On auscultation the entrance of air may be observed to be fairly free into the upper and anterior parts of the lungs, but feeble at the bases. If, however, as is often the case in adults, the condition is complicated by coexistent emphysema, the breath sounds will be generally feeble. The adventitious sounds, which especially characterise this form of the disease, are fine bubbling râles, having in a minor degree the crepitant character; they are sometimes described as 'hissing.' In severe cases they may be widely distributed over the chest, but as a rule they are most marked on the posterior aspect, and especially at the bases of the lungs. The ordinary adventitious sounds of bronchitis—sibilant and sonorous rhonchi—may also be present, chiefly over the upper and anterior aspects of the chest.

Course.—A severe attack of the kind above described may be fatal in an infant in twenty-four hours; in childhood the duration is usually more prolonged; in an adult, the subject of emphysema and addicted to alcoholic excess, we have known death to occur in four days; in the aged the disease may run a still shorter course. The indications of improvement are similar to those observed in the milder form of the disease. The duration of the attack in which recover is always prolonged, and especially so in the ts of emphysema.

ognosis.—The signs of special gravity are increasing a with lividity, delirium, and coma. Recovery in this form of the disease rarely, if ever, occurs when expectoration has ceased and the patient has sunk down in the bed, lying perhaps on one side, with the head low and bent forward.

Diagnosis.—The recognition of this type of bronchitis is often attended with great difficulty. It may simulate *pneumonia*, but its onset is not usually quite so sudden or attended with such a severe and prolonged rigor and such a marked rise of temperature as characterise that disease. Pain in the side is not so severe, whilst subjective dyspnœa is more marked. Practically, however, the differential diagnosis rests upon the results of physical examination of the chest; the absence of signs of consolidation of the lungs and the presence of bronchitic râles being the determining factors. When pneumonia occurs in an individual the subject of chronic bronchitis, with its attendant emphysema, the problem becomes more difficult. The dulness on percussion over the consolidated area may be masked, and the adventitious sounds may clearly point to an affection of the bronchi; but tubular breathing, though faint and distant, will sooner or later appear, and is evidence that something more than bronchitis is present.

Miliary tuberculosis of the lungs in children may ultimately give rise to physical signs simulating this form of bronchitis, but if the case has been carefully watched from the commencement, there should not be much difficulty in distinguishing the two affections. The onset of tuberculosis is usually insidious; there is pallor, wasting, some fever, loss of appetite, and alteration in disposition. The disease is rarely, if ever, limited to the lungs; signs of implication of the cerebral meninges or of the abdominal organs, such as enlargement of the liver and spleen, being as a rule present. If, however, the case is seen at a late stage, the resemblance to capillary bronchitis may be very close, especially when extensive broncho-pneumonia has supervened. But if expectoration is present, the results of the examination of the sputa for tubercle bacilli generally lead to a correct diagnosis.

Acute caseous tuberculosis of the broncho-pneumonic type is characterised by a higher and more continued fever, by the absence of such marked dyspnœa as occurs in capillary bronchitis, and of signs of over-distension of the right side of the heart, and by the early occurrence of mucopurulent expectoration, in which as a rule tubercle bacilli are present.

Physical examination of the chest nearly always indicates that the disease commenced at the apex of one lung, a condition practically unknown in acute bronchitis of the variety now under consideration.

The family and personal history of the patient would in this, as indeed in all other cases, require due consideration.

A first attack of *asthma* may be mistaken for bronchitis involving the smaller tubes, but the absence of fever and of characteristic cough and expectoration, with the sudden onset of dyspnœa, as a rule suffice to prevent error. It must, however, be remembered that a spasmodic element may be present in the dyspnœa of acute bronchitis.

TREATMENT OF ACUTE BRONCHITIS OF THE LARGER TUBES

The patient should at once take to bed. The temperature of the room should be between 65° and 70° F., and it will be well to moisten the air by means of steam from a bronchitis kettle. The tendency certainly is to keep the air in the room too warm and too moist, and thus to produce a condition which is very apt to increase the difficulty the patient already experiences in breathing. In a hospital ward it may be necessary to surround the bed with a tent, but this is rarely so in a private house.

The object of treatment in the early stage is to promote secretion from the inflamed bronchial mucous membrane, thereby diminishing the hyperæmia and swelling, and lessening the obstruction to the entrance of air. This is partly effected by the application of warmth to the chest. A hot linseed poultice lightly made, properly put on, and changed about every two hours, is probably the best local application; it is certainly one for which the patient nearly always feels grateful. On the other hand, a badly made, lumpy, half-warm poultice, which is never in the right position and is allowed to remain on for hours after it has become cold, is almost certainly the worst. The addition of some mustard to the first poultices may be useful, but it is important not to produce so much irritation of the skin as may prevent their continuous application. If a skilful nurse is not in attendance it is better to use stimulating liniments, containing turpentine and iodine (lin. tereb. ʒvij; tinct. iodi ʒj), as local applications, the chest being covered with a layer of cotton-wool retained in position by a light many-tailed bandage.

The drugs which are of most service in the early stage of the disease are vascular depressants, such as tartarated antimony and ipecacuanha, which give relief by increasing the secretion from the bronchial mucous membrane. Opium and morphia diminish the sensitiveness of the bronchial mucous membrane, and so lessen the cough. Pulv. ipecacuanhæ co. (gr. x–xv), which combines these two classes of remedies, is of much service in the treatment of the early stage of the disease. Liquor ammoniæ acetatis (ʒiv) and spirits of nitrous ether (♏xxx–ʒj), by acting as diaphoretics, relieve the tension of the vascular system, and indirectly the condition of hyperæmia of the bronchial mucous membrane. Formulæ in which these time-honoured remedies are variously combined are to be found in every hospital pharmacopœia. It will be sufficient, therefore, to indicate the precautions which should be observed in their use. The dose of the depressant remedies should be small (vin. antimonialis ♏xv; vin. ipecac. ♏v), and that of morphia only sufficient to allay the irritability of the bronchial mucous membrane (liq. morphinæ acet. ♏x). These drugs may be given at first every three hours, in combination with the saline mixture.

Tincture of aconite, in doses of ♏j every half-hour, may be given to fairly robust patients for eight or ten doses, or until free perspiration is produced and the temperature is lowered.

It will be well here to state clearly the limitations which govern the use of opium (or morphia) in all forms of acute bronchitis. It is quite inadmissible in the case of young children, and almost so in old people. It should never be given to procure 'a good night's rest.' It is absolutely contraindicated in the capillary form of the disease, and in all severe cases where the bronchi are extensively obstructed. A cough which is effective in getting rid of secretion should never be suppressed by opium. The drug is only admissible in the early stage of acute bronchitis of a mild type, when the cough is harassing and ineffective, and is due not to the presence of secretion in the tubes, but to the increased sensibility of the inflamed membrane. Codeia (gr. $\frac{1}{6}$), in combination with citric acid (gr. vj), vin. ipecac. (♏ij), and spirits of chloroform (♏iij), is also useful as a sedative. A saline aperient should be given at the commencement of the treatment, and a regular daily action of the bowels should be subsequently secured.

In the mild type of case now under consideration we may expect in from twenty-four to forty-eight hours to observe the change in the expectoration, which marks the transition from the stage of hyperæmia to that of free secretion. This will probably be accompanied by a fall in the temperature. Remedies which promote expectoration, such as carbonate of ammonia and senega in combination with alkalies, should now be given.

So long as fever is present, it is well to continue the use of poultices; when it has disappeared a layer of cotton-wool should be substituted.

When, at a later stage, but few catarrhal signs remain and the attack is obviously subsiding, quinine and arsenic may be given as tonics to promote convalescence.

The diet during the early febrile period should be chiefly liquid. Warm drinks, such as hot milk, beef tea, clear soup, and chicken broth, in addition to their nutritive and stimulant properties, possess that of aiding the action of the skin and of relieving the cough. Barley water, well flavoured with lemon, is useful to allay thirst. The lemon is an important addition; it appears, in the language of hospital patients, to 'cut the phlegm.' All kinds of white milk puddings may be allowed, and as soon as appetite returns, chicken and white fish may be added to the diet. Many patients, particularly men accustomed to good living, dislike intensely a liquid diet with food at short intervals; it is generally best in such cases to let them have light solid food, nearly at such times as they are accustomed to take their meals.

Stimulants are rarely necessary as such, but brandy may be given with hot water, sugar, and lemon, or with hot milk, either to produce sweating or to aid expectoration.

TREATMENT OF ACUTE BRONCHITIS OF THE SMALLER TUBES

The surroundings of the patient should be such as have been already described as regards warmth and a moist atmosphere, and jacket poultices should be continuously applied to the chest. At the onset of the disease in children a warm bath containing mustard is often of service. In adults it may be necessary to relieve the circulation by the application of leeches, six or eight in number, over the upper part of the sternum; and in strong children two or three leeches may be applied without risk of harm being done. In severe cases of rapid onset in robust adults, venesection to the amount of six or eight ounces may be required. The indications for this measure are extreme dyspnœa, cyanosis, lividity, and evidence of over-distension of the right side of the heart. Venesection is, however, rarely required in children, and should never be employed in the case of old people. If the case is seen early when the bronchial mucous membrane is still in a condition of hyperæmia, antimony, either in the form of tartar emetic (gr. $\frac{1}{3}$) or vinum antimoniale (♏xv to xx) may be given every three hours until about eight or ten doses have been taken. The action of all depressant remedies requires, however, to be carefully watched; the indications for their omission being prostration, free perspiration, and a feeble pulse. If expectoration, previously absent, should appear and the breathing become less oppressed, benefit has been derived from their use.

The dangers attendant on the administration of *opium* are tenfold greater in this than in the milder form of the disease. The patient's life practically depends upon the maintenance of the power to expectorate, and coughing is the agent by which this is effected. No treatment can therefore be worse than the administration of a drug, which deadens the sensibility of the bronchial mucous membrane, and so prevents the reflex act by which the secretion is got rid of. Patients often crave for a sleeping draught, and it may be difficult to resist their appeals; but less harm is done by the absence of sleep than by the accumulation of mucus, which must occur during sleep. A sedative draught should not be given if the dyspnœa is urgent, and there are signs of engorgement of the right side of the heart and of the bases of the lungs. Bromide of potassium (gr. xv) combined with aromatic spirits of ammonia and possibly with a small dose of chloral (gr. v to x), or in place of this half a drachm of bromidia, are the best hypnotics, if it should be considered desirable to administer one; but the period of sleep allowed must be short, say two hours, and special efforts should be made to promote expectoration when the patient is awakened.

Free purgation, by the administration of calomel (gr. iij–v) at night and a saline draught on the following morning, tends to relieve the circulation by removing any congestion of the portal vessels that may be present. It should be resorted to as soon as the case is seen, and a free action of the bowels maintained so long as evidence of engorgement of the right side of the heart remains.

Flatulent distension of the stomach and tympanites should be treated at once, the former by carminatives and purgatives, the latter by enemata of turpentine or rue; both conditions are serious, as they interfere greatly with respiration.

Stimulants may be required at an early stage of the disease; a tablespoonful or more of brandy, given with hot milk at intervals of four hours, is generally the best. All hot drinks tend to promote sweating, and long experience proves that a free action of the skin tends to relieve internal hyperæmia and congestion.

By the adoption of the foregoing measures more or less relief will be given, except in the most severe cases. Diminished viscidity of the expectoration, a lower temperature, less dyspnœa, a looser cough, and a more 'moist' character of the râles constitute the best evidence of improvement. The more active depressant remedies, such as antimony, should now be discontinued; small doses of vinum ipecacuanhæ may, however, still be given, but in combination with stimulant expectorants, such as carbonate of ammonia, tincture of squill, and infusion of senega.

When this favourable change in the general symptoms is established it is very important that it should be maintained, a so-called 'relapse' being usually due to some obvious cause. If, for example, the fire is not properly attended to during the night, the temperature of the room will fall during the early hours of the morning, and the patient will become chilled, especially if there is much moisture in the air. The same result will follow if the poultices are allowed to become cold. Inattention to the regular administration of food, stimulants, and medicine during the night and in the early hours of the morning, when there is a natural depression of the vital powers, will check the secretion and discharge of mucus. If the patient is allowed to sleep too long, or if on waking he is not sufficiently roused to expectorate, mucus will accumulate in the tubes, the breathing will be more oppressed, the temperature may rise, and he will probably be said to have had a 'relapse.' It may be mentioned here that such cases should always be under the care of trained nurses day and night; and if the wish of the friends, which is often expressed, to undertake the care of the patient during the day be acceded to, it will nearly always prove to his detriment.

It may happen that notwithstanding the adoption of the mode of treatment here laid down, or from neglect of proper treatment in the early stages of the disease, the symptoms continue to increase in gravity. This may also be due to the youth or advanced age of the patient, to feeble physique, to the presence of emphysema, or a feeble heart; or the attack may from the onset have been of the severe type which often proves fatal in spite of every care. In such cases it may be well to substitute turpentine stupes to the chest for the poultices, but they must be used with discretion, for the skin will probably by this time be tender. In children it is often desirable to stimulate respiration by a sudden affusion of a small quantity of cold water on the chest whilst the patient is in a hot bath.

In such cases also an emetic of ipecacuanha (gr. xx) may give great relief. During the act of vomiting, a large quantity of mucus is got rid of by the compression of the chest, and the breathing is for a time relieved. If its action is beneficial, the remedy may be repeated on the recurrence of the urgent symptoms. The administration of an emetic to an adult often requires some courage; if it does no good it will almost certainly do harm, and if death should shortly follow, an occurrence by no means unknown, it may possibly be attributed to the treatment and not to the disease.

In an adult, if venesection has not already been practised and there are indications of failure of the right side of the heart from over-distension, six to eight ounces of blood should be withdrawn, and the operation may be repeated if it is evident that the heart is again becoming dilated.

We have seen great benefit, which is sometimes permanent, but more often temporary, follow the inhalation of oxygen in severe cases of bronchitis. The change in the patient's aspect may be most striking, cyanosis giving place to a healthy-looking flush after the gas has been inhaled for a few minutes, and the pulse showing marked improvement in tone. Sometimes, however, the oppression of the breathing is so extreme that the patient cannot bear the face-piece of the inhaler near the mouth; in such a case it should be held, for half an hour or more at a time, as near as it can be borne; otherwise the duration of the inhalation should be about ten minutes, with intervals of half an hour or less, according to the severity of the symptoms. Before taking the apparatus into the room, it is well to make sure that you have not been supplied by mistake with an empty cylinder, and also to fit the tube on to the nozzle and turn the tap so that the slightest further turn will let the gas into the bag; otherwise the tube and bag are very likely to be blown off the nozzle, much to the alarm of the patient.

A hypodermic injection of strychnine (liq. strychninæ hydrochl. ♏ij–iij) may be given to stimulate the respiratory centre, and repeated every three hours as an adjunct to other measures.

In severe cases occurring in old people, it may be necessary to adopt from the outset a more stimulant plan of treatment, especially if the heart is feeble and expectoration difficult. As much as six or eight ounces of brandy may be required in the twenty-four hours, and a mixture containing digitalis, æther and ammonia may be given every four hours, or the digitalis (tr. digit. ♏v–x) may be added to the hot brandy and milk.

Dr. Wilson Fox was of opinion that dry râles with great dyspnœa are not infrequently due to spasm of the tubes, and advocated the use of the tincture of lobelia (♏xv–xx) in combination with ipecacuanha and ammonia, and small doses of opium or morphi in such cases. If there is no doubt as to the existence of the spasmodic element this combination of remedies may be tried, but in doubtful cases it is well to omit the opium, as a mistake might be followed by dangerous and possibly fatal results. The point may sometimes be determined by watching the effect of the inhalation of nitrite of amyl. If considerable relief to the breathing is experienced, we may be certain of the presence of a spasmodic element in the case. To observe an asthmatic paroxysm disappear completely in a few minutes when a capsule of nitrite of amyl is broken beneath the nose, and, when the effect of the drug ceases, to see the dyspnœa return as quickly as it went, is a remarkable clinical experience. The inhalation of ten to twenty minims of chloroform upon a piece of lint is also useful in similar cases, but its effect is often only temporary; the administration should not be carried farther than is necessary to relieve the difficulty of breathing.

When the severe symptoms have passed off, tonics, such as quinine, cinchona, and nux vomica, may be either added to the expectorant mixture or given separately. Iron and cod liver oil are useful at a later period. If expectoration should continue to be copious and purulent for a long period after all the acute symptoms have passed away, nourishing diet should be given and a tonic plan of treatment adopted. Tar water and remedies of the turpentine order, such as terebene, which tend to diminish secretion, are also serviceable at this period.

A change of climate is often necessary to complete convalescence; this should, speaking generally, be to one marked by a high and even temperature, in which the air is dry. All health resorts, both in this country and abroad, suitable for cases of bronchitis owe their reputation in great part to a position sheltered from cold and damp winds. This point is considered more fully in the discussion of the treatment of chronic bronchitis.

J. K. F.

CHAPTER V

CHRONIC BRONCHITIS

	PAGE
Etiology	103
Morbid anatomy	104
Symptoms	105
Physical signs	106
Course	106
Diagnosis	107
Prognosis	107
Treatment	108
Dry Catarrh. Catarrh Sec	113
Bronchorrhœa	113
Fœtid Bronchitis	114

THE term 'chronic' as applied to this form of bronchitis does not necessarily imply that its course is continuous, as in its most typical variety, viz. chronic winter cough, the symptoms are at first limited to the period when the climatic conditions are most unfavourable.

It is generally due to a recurrence of acute or subacute attacks of the disease.

Chronic bronchitis occurs in the great majority of cases in association with emphysema, of which condition it is the most common cause, and by the aid of which its permanence is secured.

Examples of the disease differ so greatly both in type and in severity as to constitute varieties which require separate notice.

Etiology.—As the most important factor in the etiology of chronic bronchitis is the occurrence of acute attacks, all the conditions which act as either predisposing or exciting causes of the acute disease may be concerned in the production of the chronic form. The presence of emphysema, of the degenerative changes incidental to advancing age, of alcoholism, chronic Bright's disease, and gout also predispose to the recurrence of attacks.

Any lesion of the cardiac valves which affects the pulmonary circulation, particularly mitral disease, by producing congestion of the bronchial mucous membrane tends to excite catarrhal changes in the bronchi. The pressure of an aneurysm of the aorta or a mediastinal growth may act in a similar manner.

Morbid Anatomy.—Emphysema is almost invariably present in those parts of the lungs which are the common sites of that change. The bases will probably be congested and œdematous, and if, as is often the case, the patient has succumbed to an acute exacerbation of the disease the lesions of acute bronchitis will be superadded to those of the chronic form.

The bronchi, especially those of medium size, contain much mucopurulent secretion, and the mucous membrane is congested and of a deep purple colour. It is often much thickened and longitudinal ridges of a greyish colour, formed by the elastic fibres of the inner fibrous coat, may be very obvious in the larger tubes. These changes cause narrowing of the lumen of the larger tubes, whilst some of those of smaller calibre may be dilated. The bronchial glands are enlarged and of a coal-black tint.

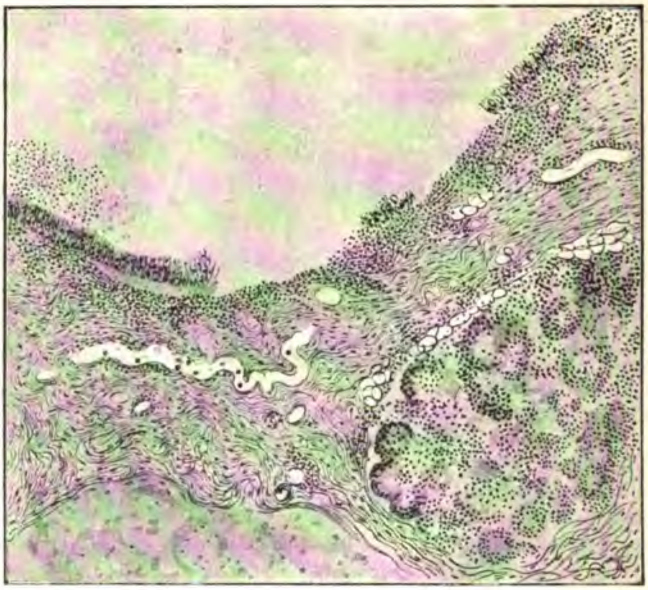


FIG. 54.—CHRONIC BRONCHITIS, SHOWING COMPLETE DISAPPEARANCE OF THE EPITHELIUM IN PLACES, AND MARKED THICKENING OF THE WALLS OF THE BRONCHUS

On *microscopical examination*, the ciliated columnar epithelium will be found to have, for the most, disappeared, its place being taken by cells of transitional form which are attached to the basement membrane. The inner fibrous coat is enormously thickened from cellular infiltration and dilatation of the blood-vessels, and its fibrous structure may be very indistinct. The muscular coat may be found in some bronchi to have undergone atrophy, in others it is hypertrophied, the difference depending probably upon the stage of the disease, atrophy succeeding hypertrophy as the cellular infiltration of the bronchial wall increases and dilatation of the tube occurs.

The cellular infiltration extends to the outer fibrous coat, and evidence of peribronchial thickening is almost always present. The cartilages may be in process of absorption, or, more rarely and especially in old people, they may be calcareous. The mucous glands may be found either filled with secretion or their secreting structure may be destroyed. The muscular coat of the small arteries is hypertrophied and the capillary vessels are dilated.

The changes in the heart and other internal organs are similar to those described under emphysema, and referred to also in the description of acute bronchitis.

Symptoms.—The mildest and most common form of the disease is that already referred to as a *chronic winter cough*; it is met with in middle-aged and elderly people, particularly in such as are somewhat obese and take a large quantity of food and alcohol. Such patients suffer from cough, perhaps preceded by some wheezing, chiefly on rising in the morning; at the same time they bring up a moderate quantity of mucopurulent expectoration, and are then, as they commonly state, 'free for the day.' There is usually some degree of dyspnœa on exertion, this being in proportion to the amount of emphysema present. So long as they can follow their ordinary occupations, and the cough is confined to the winter months, they are rarely much troubled by the condition; but, as time goes on, the summer intermission becomes shorter, and the symptoms in the winter more severe, until they are only free from cough and wheezing for a month or so throughout the year. Ultimately the cough, expectoration, and dyspnœa become continuous all the year round, but the symptoms are always more severe during periods of cold and damp weather and when fog is present.

The disease may be evolved in the manner just described, or it may occur as the result of one or more acute attacks without presenting such a prolonged course.

Cases of chronic bronchitis of a more severe type are marked by an aggravation of all the ordinary symptoms; the cough is more constant and severe, and may occur in paroxysms, during which there is marked cyanosis of the face, and after which the patient is for a time very breathless. The expectoration is less easily expelled, and when any slight exacerbation is present it may for a time be scanty and sticky in character. More often, however, it is profuse and mucopurulent, consisting of nummulated masses of a yellowish-green colour, which, according as air is or is not contained in it, sink or partially float in the liquid portion of the sputa or the sputa, if very viscid, may form a single mass in the spitting-cup.

Dyspnœa is now present even when the patient is at rest, and the breathing is laboured and accompanied by rhonchi during expiration. Very often at this period the patient begins to suffer from attacks of dyspnœa of the asthmatic type (bronchial asthma). These usually come on at night, and during their continuance his sufferings are much increased. There is also risk of the supervention of acute inflammation of the smaller tubes, a complication which, in such a condition of the lungs, is very likely to prove fatal.

A more advanced stage of chronic bronchitis is characterised by emaciation, loss of strength, night sweating, increased difficulty in expectoration, and more severe and continuous dyspnœa. The patient now lies in bed propped up by many pillows, or is unable to breathe with any degree of comfort unless sitting in a chair. There is cyanosis, the veins of the neck are distended and the fingers and toes clubbed, and all the signs of over-distension of the right side of the heart are present.

Towards the close of the case the legs become œdematous, the urine is scanty and contains albumen, tracheal râles become audible, and death occurs from dropsy due to gradual cardiac failure and 'clogging' of the bronchi with secretion which the patient is too feeble to expectorate.

Physical signs.—Much of the description given of the physical signs of acute bronchitis applies also to those of the chronic form. On inspection the signs of emphysema, including the characteristic alterations in the shape of the chest, will very likely be found. Rhonchal fremitus may be felt on palpation. The percussion note will be affected by coexistent emphysema, but otherwise will not differ from the normal. In advanced cases dulness may be present at the bases from œdema, collapse, and hypostatic pneumonia. On auscultation, if but little emphysema be present, the breath sounds will be found to be harsh, expiration being prolonged; if there be general emphysema the true respiratory murmur will be feeble, but coarse rhonchi in the larger tubes may simulate a harsh breath sound. The adventitious sounds are similar to those of the acute variety. Sibilant and sonorous rhonchi and bubbling râles predominate during an acute exacerbation or an intercurrent attack of bronchial asthma. Fine bubbling and crackling râles may be audible over the bases, but it must not be at once concluded that their presence indicates extensive œdema or bronchopneumonia, as they may be produced in the emphysematous area commonly found on the posterior aspect of both lower lobes. Râles of this character are often continuously present in the subjects of advanced emphysema. If bronchiectasis is present the hollow wavy character of the breath sounds characteristic of that condition may be audible, possibly only after the dilated tubes have been emptied by a paroxysm of cough attended with copious expectoration.

Course.—In a case of winter cough any shortening of the summer intermission marks an advance in the disease, and when the symptoms become continuous throughout the year the step is a very decided one. The occurrence of the first serious attack of nocturnal dyspnœa is also significant. Each additional pillow which the patient finds he requires to enable him to breathe in comfort in bed is evidence that dyspnœa is tending towards orthopnœa. An intercurrent acute attack may leave behind it a marked increase in the extent of the emphysema and so hasten the course of the disease.

When a sufferer from chronic bronchitis begins to lose weight the change is generally of unfavourable omen, but this is nearly always observed to accompany an acute attack and may cease when it subsides. Progressive loss of strength and increasing breathlessness on exertion are signs rarely unobserved or misinterpreted by the patient. Clear evidence of the occurrence and continuance of tricuspid regurgitation is an indication of cardiac dilatation and degeneration, and marks a decided advance in the morbid processes incidental to the disease.

Diagnosis.—In all cases of chronic pulmonary disease the sputum should be examined for tubercle bacilli, not once only but frequently. If this precaution is not adopted, cases in which general or compensatory emphysema, the result of arrested or quiescent tuberculosis, is followed by chronic bronchitis, are liable to be misunderstood. But in the fibroid form of tuberculosis, which is usually present in such cases, tubercle bacilli are frequently absent from the sputa. Pulmonary tuberculosis may also be engrafted upon chronic bronchitis and emphysema, and its recognition may be difficult without the aid derived from an examination of the sputum.

Whenever the signs are more marked at the apices of the lungs, or at one apex, than elsewhere, tuberculosis should be suspected; this holds good even though the signs are wholly catarrhal in character, and tubercle bacilli are not found in the sputa.

In a case lately under observation, in which loud sonorous rhonchi were audible over both lungs, the diagnosis of chronic bronchitis and emphysema was proved to be, at any rate, very incomplete by the discovery of dulness over the manubrium, and other signs indicating compression of the trachea by an aneurysm of the transverse part of the arch of the aorta. In another case admitted into the Brompton Hospital, under the care of the writer and diagnosed by him as one of bronchitis and emphysema, a small growth originating in the mediastinal glands and extending along the main bronchi was found on post-mortem examination. The absence of dulness on percussion may render the diagnosis of such a case very difficult.

Stenosis of the trachea or main bronchi, the result of the contraction of syphilitic gummata, is very likely to cause symptoms which may suggest a diagnosis of chronic bronchitis.

Prognosis.—A sufferer from chronic winter cough who is able to spend the cold months of the year in a suitable climate may fairly hope to escape the annual recurrence of his malady, but whatever degree of emphysema may be present will necessarily remain. In such patients as continue in this country and attempt to follow their usual occupations the disease will almost certainly progress.

attack. The relief to the sense of tightness within the chest, as soon as free perspiration is induced, is described as marvellous. The Turkish bath should be followed by a warm bath and retirement to a well-warmed bed, and a respirator with terebene (♏xx–xxx) on the sponge should be worn during the night. This mode of treatment is certainly worthy of an extensive trial.

When the acute stage is passing off, squill may be administered in combination with carbonate of ammonia or the compound pill of ipecacuanha and squill may be given at bedtime.

Tonic remedies such as perchloride of iron, cinchona, strychnine, and cod-liver oil should be given during convalescence from an intercurrent acute attack or in periods of comparative freedom from urgent symptoms. They are also of much service in checking profuse secretion.

The attention of the sufferer from chronic bronchitis is, as a rule, chiefly centred upon the cough and he is urgent for its relief. Treatment should, however, be directed more towards the cause of the reflex act and less to the act itself, which is merely evidence of an irritable condition of the bronchial mucous membrane, due to the loss of its protective epithelial lining or of the presence of secretion within the tubes. As a general rule greater relief to the cough is obtained from the use of remedies which promote expectoration and of those which diminish secretion than from such as have a merely sedative action. A constant harassing and ineffective cough may be relieved by the use of sedative remedies, but a necessary and effective cough should not be checked, as the secretion must be got rid of and the cough is the only agent for this purpose. The cautions already given as to the use of sedatives and particularly opiates in the treatment of acute bronchitis are to be observed (*vide* p. 99). The inhalation of the vapour arising from a pint of boiling water to which a teaspoonful of the compound tincture of benzoin has been added may afford relief to an irritable mucous membrane, or a small dose of tinct. camphoræ comp. (♏x–xv) may be added to the expectorant mixture. The inhalation of a few drops (♏x–xx) of chloroform is often of use in checking a very irritating cough. Codeia (gr. $\frac{1}{6}$) in combination with citric acid (gr. vi), spirits of chloroform, and mucilage may be given in cases where more decided sedatives are inadmissible. The warm alkaline draughts already recommended are also of great service in relieving cough. Barley water well flavoured with lemon is a pleasant drink and certainly has a similar effect. If the state of breathing allows the patient to wear an oro-nasal respirator, ten drops of a 20 per cent. solution of menthol in alcohol may be poured on the sponge, and the instrument worn for some hours at a time.

The condition of the bronchial mucous membrane may be favourably influenced by the use of remedies which are in part eliminated through the air passages. Most of the drugs of this class are given with a view to diminish excessive secretion; such are turpentine, terebene (♏v–xx), terpene (gr. ij–vj), creasote, tar, copaiba, benzoin, ammoniacum, and tolu. Of these, turpentine and terebene are the most valuable. In common with nearly all the remedies just mentioned, they may be either administered internally, preferably in capsules (turpentine ♏x, terebene ♏v), or used as an inhalation (*e.g.* ten drops of a mixture of equal parts of terebene, carbolic acid, and spirits of chloroform), or both methods of use may be combined. The perforated zinc respirator suggested by Dr. Burney Yeo is a very convenient means of employing many of these remedies by inhalation.

Tar, in the form of tar water or pills, is a remedy long employed in checking the profuse secretion of chronic bronchitis.

Creasote may be given either in capsules, or *perles* (each containing from two to three minims), or by inhalation.

Chloride of ammonium, in the form of a spray, is also of great use, and may be combined with turpentine or its derivatives. The use of a spray of ipecacuanha wine has been strongly advocated by Drs. Ringer and Murrell.

When a case is first seen in the advanced stage of which urgent dyspnœa, cyanosis, and over-distension of the right side of the heart are the prominent signs, venesection to the extent of six or eight ounces may give great temporary relief. Inhalations of oxygen should also be used, and subcutaneous injections of strychnine (gr. $\frac{1}{30}$–$\frac{1}{20}$). A mixture containing ether, ammonia, and digitalis may also be given every four hours.

Turpentine stupes should be applied to the back and front of the chest alternately, and their use continued so long as they can be borne without great discomfort. Emetics are probably less employed now than formerly in the treatment of severe cases of chronic bronchitis in adults, when secretion is profuse and is tending to accumulate and block the tubes. It requires a nice discrimination to determine whether the strength of the patient is sufficient to withstand the depressing effect of an emetic. If in doubt on this point it is better to avoid its use. If an emetic is given, great temporary relief may be afforded by the expulsion of a large quantity of mucus during the act of vomiting.

Stimulants, of which good brandy is probably the best, are generally required in severe cases; the dose must be determined with reference to the age and general condition of the patient and the state of the pulse and respiration.

The *diet* should be 'plain' and easily digestible, and all articles likely to give rise to flatulent distension of the stomach, and dried and salted foods which tend to increase the cough, should be avoided.

In the treatment of an attack of dyspnœa of the asthmatic type occurring during the course of chronic bronchitis, the drugs of greatest value are iodide of potassium in large doses (gr. viij–xv), extract of stramonium (gr. $\frac{1}{4}$) and the ethereal tincture of lobelia (♏xx) in combination with stimulant expectorants, such as carbonate of ammonia (gr. iij to v) or ether. Morphia, which is of such great service in the purely spasmodic type of asthma, should, if employed in these cases, be used with the greatest possible caution. A free action of the bowels, to be obtained by the use of saline purgatives, tends to relieve the congestion of the bronchial vessels, and is an essential aid to other methods of treatment.

The use of *compressed air baths* in the treatment of emphysema and of chronic bronchitis may be attended with very beneficial results. We have given the method an extensive trial at the Brompton Hospital, and our opinion agrees with that of Dr. Theodore Williams ('Aero-therapeutics,' p. 106), who speaks decidedly in its favour. Patients almost invariably state that whilst in the bath they breathe with less difficulty, and in some cases permanent benefit results. On the Continent, where this method of treatment is more extensively employed, it is held in higher esteem than in this country, where the number of baths available is limited. The subject is more fully discussed in the chapter on Emphysema (p. 177).

Climatic treatment.—The most desirable conditions are an equable temperature, warmth, sunshine, and absence of cold winds and fog, a combination not easily obtainable in this country. All those who can afford it and are able to do so should certainly pass the winter months abroad, where a large field of choice as to quarters lies open to them.

Many sufferers habitually winter on the Riviera, the most sheltered place being Mentone. The disadvantages of the climate are the great difference between the sun and shade temperature and the prevalence at certain periods of winds which come from almost every quarter of the compass, each having some particularly unpleasant character of its own. These conditions obviously render it necessary for the patient to exercise the greatest prudence in avoiding exposure to cold. For some cases a still drier climate such as that of Egypt is found to be more suitable.

Madeira is, from a purely climatic point of view, preferable as a winter resort for cases of chronic bronchitis. The moist air allays the irritability of the bronchial membrane, and so diminishes cough, whilst the equable temperature lessens the risk of fresh chill.

Those who are unable to leave for a longer period may derive considerable benefit from a stay of a fortnight or three weeks at Madeira about Christmas time. Many patients, after a short stay, find the climate of Madeira too relaxing, and betake themselves to the Canaries, where the air is drier and more bracing.

Algiers, Ajaccio, and Corfu are also much frequented by catarrhal subjects during the winter months.

In this country the most sheltered places in which the sufferer from chronic bronchitis can spend the winter months are Torquay, Falmouth, Penzance, Bournemouth, and the towns along the Undercliff of the Isle of Wight.

Sufferers from chronic bronchitis often derive much benefit from a course of muriated alkaline waters (Ems, Royat), or of sulphur waters (Eaux Bonnes), or of weak arsenical waters (La Bourboule or Mont Dore), followed by a residence at some sheltered place in the Black Forest or elsewhere.

DRY CATARRH (CATARRH SEC OF LAENNEC)

This affection is described by Laennec as of extremely common occurrence, and as affecting 'almost all the inhabitants of cold sea coasts and humid valleys,' and 'one half at least of the most healthy adults in the driest parts of France.' It is said to be due to chronic congestion of the mucous membrane, particularly of the smaller bronchi, and to be characterised by the expectoration of semi-transparent airless globules of a pearly grey colour, 'of the consistence of pitch, and about the size of a hemp or millet seed.' The respiratory murmur over the part affected is said to be entirely or almost completely absent. The affection is described as remaining moderate in degree and perfectly latent for a long series of years, the only sign being slight dyspnœa on attempting to run or ascend a height. Later on, dyspnœa is felt even when at rest, and, later still, 'the attacks of oppression are sufficiently severe to merit the name of *asthma*, and they usually last several days.' It is true that dry catarrhal sounds are not uncommonly present in the bronchi in the subjects of emphysema, and that attacks of asthma and bronchial asthma often terminate with the expectoration of pearly masses similar to those described by Laennec, but apart from these common conditions we know of no affection having the characters above described, nor do we think it probable that complete absence of the respiratory murmur over extensive areas of the lungs is ever due merely to engorgement of the bronchial mucous membrane.

Bronchial catarrh accompanied by very little secretion is not uncommon in gouty subjects, and reference will be found to it in the description of secondary bronchitis of gouty origin (*vide* p. 116).

BRONCHORRHŒA—*syn.* BRONCHORRHŒA SEROSA (BIERMER), CHRONIC IDIOPATHIC PITUITOUS CATARRH (LAENNEC), BRONCHO-BLENNORRHŒA, MUCOID ASTHMA

Under the heading 'chronic idiopathic pituitous catarrh,' Laennec described an affection, characterised by paroxysms of cough and expectoration usually occurring night and morning, the quantity of sputa on each occasion being possibly as much as two or three pounds. It is predisposed to by 'frequent renewals of acute mucous catarrh' (acute bronchitis) and attacks the 'aged or adults past the prime of life,' and gouty subjects. Laennec relates two cases, one that of a man upwards of seventy years of age who had expectorated daily for ten or twelve years about four pounds of a colourless ropy and frothy liquid; the other that of a man over sixty years of age, of fair general health, able to take walking exercise, who had brought up every morning, 'by easy vomiting repeated at short intervals for some hours, from three to six pounds of a liquid perfectly resembling the white of egg mixed with one third part of water.'

Andral describes two cases of a similar character which ran a more rapid course, and in which, on post-mortem examination, no other lesion or cause of death was discovered.

Cases of chronic bronchitis and bronchiectasis with profuse expectoration are not uncommon, but those here described present unusual features, and that, in an idiopathic form, they are rare is admitted by Laennec. He states, however, that 'this affection is seen in a high degree of development when a great number of miliary tubercles are simultaneously formed in the lungs and remain long in the same state.'

FŒTID BRONCHITIS (PUTRID BRONCHITIS)

This variety of the disease is very closely associated with bronchiectasis; but fœtor of the expectoration, which is the most characteristic feature of the affection, may accompany acute or chronic bronchitis without dilatation of the bronchi. Such cases, however, are of rare occurrence, and in any case it must be extremely difficult to exclude the possibility of bronchial dilatation. The features of putrid bronchitis, as regards the character of the sputa, the occurrence of pyrexia in association with broncho-pneumonia and gangrene, and the course of the disease, agree so nearly with the description of bronchiectasis given in this work, that we must refer our readers to the chapter on that subject. The only treatment of bronchitis attended by fœtid expectoration, whether accompanied by bronchial dilatation or not, which is likely to be successful is the use of the creasote vapour bath (*vide* p. 138).

J. K. F.

CHAPTER VI

SECONDARY BRONCHITIS

PAGE
a. In acute febrile diseases 115
b. In gout 116
c. In tuberculosis 117
d. In syphilis 117
Treatment of secondary bronchitis 117

BRONCHITIS occurs as a complication of so many morbid conditions that it requires separate notice under the above heading.

In most of these cases, however, the variations from the normal type as regards symptoms and physical signs are too slight to need detailed description, and it will be sufficient to indicate the relative importance of the several diseases as factors in the etiology of the affection, and the influence which they have upon its treatment.

a. **Acute febrile diseases.**—In measles and enteric fever the bronchial complication assumes the greatest importance. It may, indeed, in both cases be the immediate cause of death. In the former, during the pyrexial period, the smaller tubes are very liable to be involved, and the inflammation may spread to the alveoli, producing broncho-pneumonia. In the latter there may be some râles at an early period of the disease, but it is not as a rule until the third week that the condition of the lungs gives cause for anxiety. The breathing then becomes quickened, the face dusky, and bubbling and crackling râles are found at the bases of the lungs; but cough and expectoration may be absent, an important point in the differential diagnosis of typhoid fever and bronchitis. It is probable that the pulmonary complications of typhoid fever are due to the presence in the vessels of the lungs of the specific virus of the disease, and are not the result of an ordinary bronchitis extending to the alveoli.

As a complication of typhus fever, bronchitis is almost always found in association with hypostatic congestion of the bases of the lungs, and the exudation into the tubes is probably, in part at least, due to congestion of the vessels, and not to an acute inflammation of the lining membrane of the bronchi.

Bronchitis, with collapse of portions of the lungs, may occur as a sequel of whooping cough, and bronchial catarrh during convalescence may cause a return of the characteristic symptoms of the original disease.

Bronchitis may also be met with as a complication of smallpox, scarlatina, and some other acute diseases, but in such it rarely assumes importance.

The bronchial and pulmonary complications of influenza are described elsewhere (*vide* p. 530).

b. **Gouty bronchitis.**—It is in this variety of the disease that the influence of the etiological factor is most distinctly shown, and its recognition is essential for successful treatment.

In individuals who have suffered from well-marked attacks of arthritis, there is as a rule but little difficulty in recognising the presence of the gouty element; but prior to such an occurrence, and also in the subjects of hereditary and what is termed 'suppressed' gout who have never had any typical manifestation of the disease, its influence in the production of a bronchial catarrh may not be so obvious.

The affection in its milder form is usually found in association with emphysema, in men who have reached or passed middle age. It is marked by the presence of a 'dry catarrh' (catarrh sec of Laennec), if such an expression is permissible. Cough is the most prominent symptom, and is in its violence quite out of proportion to the small amount of expectoration by which it is attended; it is not infrequently accompanied by a degree of congestion of the face, almost amounting to cyanosis. The sputa are glutinous and of the so-called 'pearly' character. Sibilant rhonchi are present, and the breathing may be accompanied by 'wheezing' at night or in the early morning. The symptoms, owing to the co-existence of emphysema, may be more prominent during the winter months. Sufferers from gouty bronchitis are often subject to attacks which they have the support of the late Dr. Murchison in attributing to functional derangement of the liver. These are characterised by depression of spirits, pain in the right hypochondrium, anorexia, furred tongue, constipation, and the passage of dark-coloured, highly acid urine, in which on cooling there is an abundant deposit of pink lithates.

During the continuance of such an attack the bronchial symptoms are generally more pronounced.

Another form of gouty bronchitis met with in middle-aged persons is characterised by a somewhat sudden onset and far more severe symptoms. The special feature which it presents, in addition to those of the milder form, is marked functional disturbance of the action of the heart, in which respect it resembles many other manifestations of lithæmia or so-called 'latent' gout. Such attacks may be characterised by an almost sudden onset and signs of severe pulmonary congestion, such as dyspnœa, cough, and the presence of fine bubbling and crackling râles, particularly at the bases of the lungs; but pyrexia may be very slight or even absent. These symptoms may be followed by an attack of acute arthritis, or they may subside under treatment without the appearance of any joint affection. Some refuse to recognise them as 'gouty' unless they are accompanied by the most complete manifestation of that disease, viz. acute arthritis; but this is akin to denying the connection between a bud and a flower in full bloom.

The subjects of gouty bronchitis may subsequently develop chronic interstitial nephritis; and under such circumstances attacks similar to those just described may occur from temporary cardiac failure and pulmonary œdema.

c. **Catarrh of the bronchi**, occurring in the subjects of **tuberculosis** of the lungs or in those inheriting a tendency to tubercular disease, does not present features which are so distinctive as to require separate description.

In many cases of pulmonary tuberculosis there is no marked tendency to bronchial catarrh, but catarrhal symptoms are usually present when tuberculosis is associated with emphysema, a condition of more frequent occurrence than is generally believed.

d. **Syphilitic bronchitis** is considered in the chapter on Pulmonary Syphilis (*vide* p. 449).

TREATMENT.—Bronchitis complicating the acute specific diseases requires, as a rule, a stimulant plan of treatment, as cases in which the affection is of sufficient importance to need direct attention are usually of a severe character and are often associated with some degree of cardiac failure. Carbonate of ammonia, or aromatic spirits of ammonia and spirits of æther may be combined with digitalis, the use of the latter being determined by the condition of the heart.

Inhalations of oxygen and hypodermic injections of strychnine may also be required.

Stimulating applications to the chest are generally necessary.

The milder form of gouty bronchitis is best treated by careful attention to diet and the avoidance of chills. So long as the patient, by indulgence in alcohol and a highly nitrogenised diet, maintains a high tension within his vessels the cough will probably continue, whereas abstinence from alcohol and a 'white diet' of fish, chicken &c., suitable to the gouty state will almost certainly by itself give relief. Warm alkaline drinks, as recommended in the treatment of acute bronchitis, are specially useful in these cases; they may be given at bedtime and before rising in the morning. Free action of the bowels should be maintained by the use of saline aperients, such as Carlsbad salts.

For more serious attacks of gouty bronchitis a more active treatment is necessary. Free purgation by means of salines and mercurials, and the administration of the iodides and bicarbonates of potassium or sodium fulfil the most important indications. Colchicum may be added with advantage unless there is marked failure of strength. The intermittence and functional disturbance of the action of the heart is to be met by the elimination of the waste nitrogenous products and the lowering of arterial tension rather than by the use of direct cardiac tonics, such as digitalis.

Stimulants may be required, but are to be avoided unless urgently called for.

More robust patients may be recommended to take a course of waters at Carlsbad, Marienbad, Homburg, or Kissingen. The more weakly sufferers from this form of bronchitis resort with advantage to Ems, La Bourboule, Eaux Bonnes, and elsewhere on the Continent for treatment by the inhalation of pulverised or atomised spray and drinking of alkaline waters.

It is obvious that those prone from their parentage to tuberculosis should avoid all conditions likely to diminish the functional integrity of the lungs, amongst which repeated catarrh of the bronchi must certainly be numbered. That there are such persons we continue to believe, in spite of recent confident assertions to the contrary.

In bronchitis occurring in a tuberculous subject, cod-liver oil is of great use, and a general tonic plan of treatment is indicated.

J. K. F.

CHAPTER VII

PLASTIC BRONCHITIS

	PAGE
Pathology and morbid anatomy	119
Etiology	120
Symptoms	121
Physical signs and diagnosis	122
Prognosis	122
Treatment	122

A DISEASE of rare occurrence, characterised by the exudation of a plastic fibrinous material which forms either solid casts or hollow cylinders within the bronchi.

Pathology and morbid anatomy.—Nothing definite is known as to the pathology of this affection. The appearance of the exudation naturally led to the suggestion that the disease is a form of diphtheria involving the bronchi, but in their clinical course the two diseases do not resemble one another, and there are practically no other facts which lend support to this view. That plastic bronchitis is due to the presence of a micro-organism is possible, and cultivation and staining experiments may eventually throw light upon its pathology. The exudation may be localised to a few tubes only, or may be found in numerous areas scattered through both lungs.

In some cases the casts expectorated from day to day resemble each other so closely as to prove clearly that they are formed again and again in the same bronchus.

Nearly all pathological museums contain specimens of fibrinous casts expectorated by patients suffering from the disease, but preparations showing the casts *in situ* are very rare. This might have been expected, as a fatal termination is unusual, and when it occurs no casts may be present in the tubes after death, although they have been freely expectorated during life.

The casts vary in size according to the diameter of the bronchus in which they are formed, but, like those found in 'massive pneumonia,' they do not completely fill the tubes. The smaller are usually solid, the larger may be hollow cylinders. They are often much branched, ending in fine rootlike processes (*vide* fig. 55), corresponding to the minute subdivisions of the bronchial analogy it is probable that there is some definite pulmonary lesion to account for it, as blood in large quantity does not come from the vessels of the bronchial wall. A case marked by the recurrence at varying intervals of attacks, each consisting of a succession of paroxysms similar to those above described, is by some writers classed as 'chronic.' The subdivision, however, of cases into 'acute' and 'chronic,' based chiefly upon the fact of recurrence, which is a characteristic of the disease, is quite arbitrary and of little practical value. A single attack entails a liability to the disease which may last for life, but the intervals may be very prolonged, as is shown by a case in which the disease appeared at the following ages: 36, 41, 42, and 60 years.

Physical signs and diagnosis.—The disease does not appear to have been recognised in any case prior to the expectoration or dislodgment of the casts. When the diagnosis has been made it may be possible to determine the site of their formation by the presence of catarrhal signs limited to a certain area, followed possibly by evidence of complete bronchial obstruction and pulmonary collapse in the same region. The expectoration of the cast may be marked by the return of the breath sounds, and the percussion note, previously dull, may, as the lung re-expands, become resonant; but dulness, if due to pneumonia, would of course not suddenly disappear. In any case of paroxysmal dyspnœa without obvious cause, suddenly relieved by expectoration, a careful examination of the sputa should be made, as the cast may be rolled up into a rounded mass and so thickly covered with mucus as to make its recognition difficult until it has been floated in water.

Prognosis.—The marked liability to recurrence necessarily requires to be borne in mind, but a fatal termination is rare in cases uncomplicated by definite pulmonary disease, such as tuberculosis. In a case recorded by Dr. Fagge the patient died suddenly from asphyxia, owing to the lodgment of a displaced cast at the bifurcation of the trachea.

Treatment.—The only drug which appears to have any effect upon the disease is iodide of potassium in large doses; it is said to have been used with complete success in some cases, and is certainly worthy of trial. In a case recently under the observation of one of our colleagues, it was thought that intratracheal injections of glycerine—a teaspoonful at a time—given several times daily, aided the separation of the casts. We should now be disposed to try the continuous inhalation of creasote vapour, if a suitable case presented itself.

J. K. F.

CHAPTER VIII

BRONCHIECTASIS

(DILATATION OF THE BRONCHI)

PAGE
Etiology 123
Morbid anatomy 125
Mode of production 128
  Intrinsic causes 128
  Extrinsic causes 129
Clinical varieties 131
Symptoms 131
Course 133
Physical examination 134
Diagnosis 135
Prognosis 137
Treatment 137

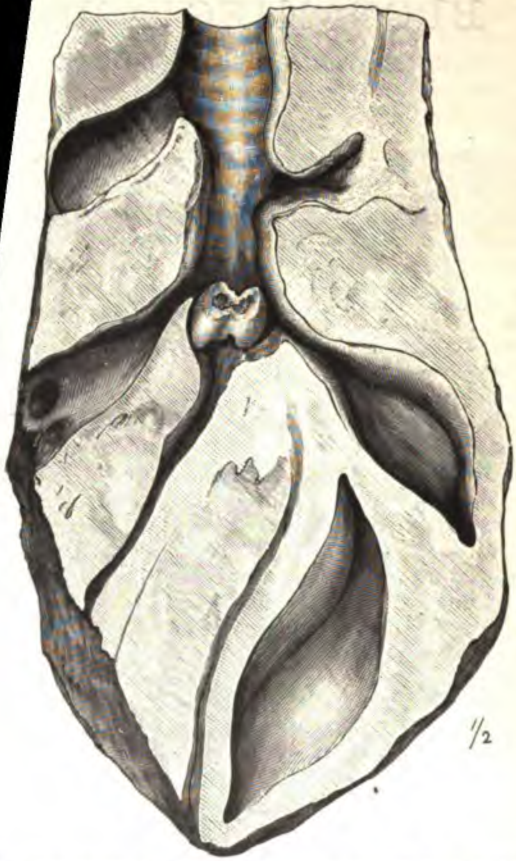
This affection was observed in 1808 by Professor Cayol, then a student in Paris. He brought his specimens under the notice of Laennec, to whom medicine is indebted for the first clinical and pathological description of the disease.

Dilatation of the bronchi, viewed from the standpoint of morbid anatomy, is a condition of common occurrence, as it is frequently found in association with pulmonary tuberculosis. From a clinical point of view, however, the affection is comparatively rare.

Etiology.—Dilatation of the bronchi is rarely, if ever, a primary disease; it may be traced to some preceding affection of the bronchial wall or of the lung or pleura. The post-mortem records of the Brompton Hospital from 1887 to 1894 contain reports of thirty-five cases in which the condition was present in a marked degree; an analysis of these cases shows them to have originated thus:

	No. of cases
Acute bronchitis	1
Chronic bronchitis	7
Syphilitic stenosis of the bronchi	1
Pressure on bronchi by a mediastinal growth or an aneurysm	1
Foreign bodies in the bronchi	2
Bronchitis and asthma	1
Broncho-pneumonia	5
Lobar pneumonia	2
Chronic pneumonia and cirrhosis of lung	5
Pulmonary tuberculosis	4
Pleurisy and empyema	3
Hydatid cyst of the kidney	1
Spinal curvature	2
	35

istrates the common causes of the affection. As to
quency of their occurrence it is difficult to make any
ent, but it is probable that this is fairly indicated by
cases here attributed to the various causes. We
l with one case in which localised bronchiectasis



. 56.—CYLINDRICAL BRONCHIECTASIS SECONDARY TO THE LODGMENT OF A TOOTH IN A LARGE TUBE

l upon the absorption of the blood in a hæmorrhagic in-
l.

.—Most statistics show a considerable preponderance of
e sex. In the above-mentioned cases twenty-nine occurred
s and six in females.

.—The affection may be met with from childhood to old
t it is most common in middle life. Excluding two cases

which occurred in children aged six and nine, and one in a man of seventy-three years, the average age was thirty-four years.

The above, however, refers to the age at death, and is a statement of comparatively little value, as the affection often runs a very chronic course, many cases originating in childhood and lasting twenty, thirty, or even forty years. The varied etiology of the condition shows of how little importance the age factor must necessarily be.

Morbid anatomy. *Varieties.*—Two chief varieties of the affection are met with, the one *cylindrical*, *simple*, or *uniform*, the other *saccular* or *globular*. When the dilatations in the cylindrical form taper towards their extremities, the term *fusiform* is often applied. In another form which is occasionally observed beadlike dilatations are present at intervals upon a bronchus, the lumen of which is not elsewhere enlarged; this has been named *moniliform*.

In advanced cases the cylindrical and sacculated forms are often found in association.

Cylindrical form.—This occurs chiefly in the larger tubes and those of medium calibre (*vide* fig. 56), but it may extend from the root to the surface of the lung. A dilated tube is rarely of uniform diameter for any considerable distance; it usually presents constrictions here and there in its course, and tends to undergo a secondary enlargement at its distal end. The constrictions are often due to a new growth of fibrous tissue which has replaced the muscular coats, and are sometimes so distinct as to form incomplete septa.

Saccular form.—The appearances presented by the most typical examples of this form of the affection (*vide* fig. 57) are in marked contrast to those just described. On section of the lung all trace of pulmonary tissue may have disappeared over areas of considerable extent, and, in its place, large numbers of rounded, smooth-walled, shiny saccules are seen, each presenting a small opening in its floor. Their walls are usually thinner than normal, and neighbouring spaces may communicate. A whole lobe may be thus affected, or in some cases the whole lung, constituting the condition known as 'turtle lung,' from its similarity in appearance to the lung of that animal. At other parts of a specimen which shows the advanced condition earlier stages of the change may be seen, and the saccules there will be much smaller, and separated by areas of lung tissue in which the tubes are not dilated. In rare cases a single large saccule is found. The terminal bronchi are usually first affected, and the distal end of the dilated tube is generally closed at an early stage of the process.

Size.—The size of the dilatation necessarily varies with that of the affected tube, but is said to be proportionately greater in the case of the smaller tubes (Biermer). A cylindrical dilatation of a large tube which will admit the forefinger is by no means rare. Saccular dilatations in the early stage are so small as only to be discoverable by the aid of the microscope, whilst in the most advanced stage they may even be equal in size to a hen's egg.

Extent.—In the great majority of cases a large number of tubes are found (post mortem) to be affected, as is shown by the fact that, in the thirty-five cases above referred to, dilated bronchi were found in all the lobes of one or both lungs in twenty-seven, whereas the lesion was limited to one lobe in only eight. In twenty-three cases (65 per cent.) both lungs were affected.

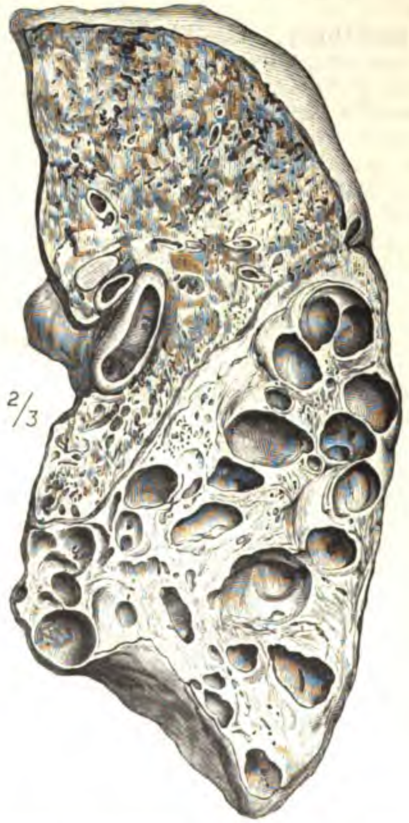


FIG. 57.—SACCULAR BRONCHIECTASIS OF THE LOWER LOBE OF A LUNG

At its commencement, however, the affection is much more limited. Frequent cough, the inhalation of secretion, and the intercurrent attacks of bronchitis and septic broncho-pneumonia, which at one period or another are almost sure to occur, combine to favour its spread. Speaking generally, it may be stated that when the condition follows acute or chronic bronchitis or broncho-pneumonia, especially in childhood, many tubes are dilated; whereas, when it is a sequel of collapse from pleural effusion, lobar pneumonia, the impaction of a foreign body, or stenosis of a large bronchus, it may for a time be limited to the area of lung affected by the primary lesion. The lower lobe is more often primarily affected, but, when the lesion is secondary to tuberculosis, the apex is usually its earliest site.

Changes in dilated bronchi.—In some cases the mucous membrane only shows the lesions of acute inflammation, but when the larger tubes are affected it is often swollen and reddened, and presents a velvety appearance. In the more chronic cases it may be greyish in tint and granular on the surface. The fibrous structures of the bronchial wall are usually in such cases thickened, and fibrous peri-bronchitis may be present, the thickened tubes on transverse section standing out from the cut surface of the lung; but in other cases, particularly in those of the sacculated variety, the wall of the tube may have undergone decided thinning. The tubes may be empty, or may contain large quantities of thick purulent fluid of remarkably fœtid odour. This may be of a dirty grey or greyish-pink colour, stringy, curdy, or inspissated. In other cases the dilated tube throughout its length may be found filled with clear, sweet, gelatinous-looking mucus, somewhat resembling the contents of a culture tube of nutrient gelatine. On closer inspection in such a case it will be seen that the upper surface presents a yellowish tinge, and here a growth of micro-organisms may be found, rendering the likeness still more complete. In cases where all communication from the air has been shut off, cystlike cavities may be found filled with caseous-looking material.

Changes in adjacent lung tissue.—The condition of the lung in the immediate neighbourhood of the dilatation varies greatly, and, as a rule, no single lesion is present alone. The lung may be healthy, emphysematous, collapsed, condensed, or fibroid. It is frequently œdematous. A lobe, or even the whole lung when the change is extensive, may be consolidated, tough, iron-grey in tint, and densely indurated.

Extensive areas of broncho-pneumonia and of gangrene may also be found around dilated tubes.

Associated pathological lesions.—In addition to the changes found in the affected tubes, more or less general bronchitis is usually present, the mucous membrane being often intensely red or of a purplish tint. Acute pleurisy with effusion is rarely met with, but pleural adhesions are generally present post-mortem, though they have been absent in many cases submitted to operation. The adherent pleura is often much thickened, and the interlobar septa appear as dense fibrous bands extending from the surface towards the root of the lung. The interlobular connective tissue may also be increased. The process by which the thickening of the pleura and of its prolongations between the lobes is effected will be discussed later.

Perforation of the pleura and pneumothorax may occur, or the cavity originating in a dilated bronchus may even penetrate the chest wall, and either rupture externally or form an abscess beneath the muscles. Perforation into the cellular tissue of the mediasti may give rise to interstitial or subcutaneous emphysema; but all these events are of very rare occurrence, and when met with are usually complications of gangrene.

In the cases accompanied by emphysema the morbid conditions incidental to that disease will be present. Of these, hypertrophy and dilatation of the right side of the heart and congestion of the systemic veins are the most important. Long-continued profuse purulent discharge may induce lardaceous disease.

Cerebral abscess was the immediate cause of death in four of the series of cases previously referred to.

The kidneys may present indurative changes from chronic venous congestion, or they may be lardaceous. Chronic parenchymatous nephritis has also been observed.

MODE OF PRODUCTION

This is a point on which considerable difference of opinion exists; all writers, however, agree that dilatation of the bronchi is not a primary disease, but a secondary result of some lesion, involving either the bronchial tubes, the lung, or the pleura. It will therefore be convenient to consider the mode of production under the headings *intrinsic* and *extrinsic*.

INTRINSIC CAUSES

(1) **Acute and chronic bronchitis.**—As dilatation of the bronchi may occur in the absence of any lesion of the surrounding lung tissue, it is clear that the changes found in the bronchial walls are the only pathological conditions absolutely necessary for its production. No explanation, therefore, which does not take account of this fact can be accepted as satisfactory. Chronic bronchitis is, as we have seen, in the great majority of cases associated with emphysema, and not with bronchiectasis. Violent expiratory efforts with the glottis closed, or partially closed, will give rise to emphysema, and for the uncomplicated cases of bronchiectasis, just referred to, a similar explanation is the only one which meets the facts. It is not easy to say why in the one case the alveoli dilate and in the other the bronchi, but it probably depends upon the fact that, when bronchiectasis occurs, inflammatory changes in the bronchial wall have led to a loss of elasticity and muscular tone, and possibly to atrophy of the muscular tissue; conditions most favourable to the occurrence of dilatation. Violent cough probably under all circumstances causes a slight temporary dilatation of the bronchi, but this is immediately followed by recoil so long as the integrity of the elastic and muscular tissues is maintained. If, however, these tissues be weakened and their loss of elasticity be out of proportion to that of the lung, the result will be bronchial dilatation instead of emphysema. Retained secretion, which is no longer permeable to air, must form a better *point d'appui* for such a force than air contained in the tubes, and secretion may be retained before the lung tissue beyond has undergone collapse. A deficient

entry of air into any part of the lung, by producing incomplete expansion of the alveoli and so defective support of the bronchi, would necessarily favour dilatation of the tubes; but such a condition, although commonly present in cases of bronchiectasis, cannot be regarded as essential.

(2) **Bronchial stenosis.**—This is an important factor in the etiology of bronchiectasis. Narrowing may be caused by the contraction of gummatous tissue in the bronchial wall, by the pressure of an aneurysm or a morbid growth, or the presence of a foreign body, and in other ways; but, however produced, it leads to dilatation of the tubes beyond the site of constriction. Various causes contribute to this result.

(*a*) Accumulation of secretion in the tubes leads to obstruction of the bronchioles and collapse of the tributary area.

(*b*) Changes in the bronchi and peribronchial tissues diminish the power of resistance to pressure from within.

(*c*) Fibrous thickening of the interlobular and interalveolar connective tissue, and of the lung as a result of inflammation, lead to contraction of the surrounding tissue.

The above conditions render effective the dilating force of violent expiratory efforts with a partially closed glottis, and this we regard as the indispensable factor in the production of dilatation.

(3) **Obstruction of a bronchus.**—When a bronchus is suddenly obstructed, as, for instance, by a foreign body which fills its lumen, collapse of the area of lung to which it is distributed is the usual result, and this is followed either by emphysema of the surrounding tissue or by dilatation of a neighbouring bronchus. When, however, complete obstruction follows gradual narrowing, the tubes beyond have usually already undergone dilatation.

EXTRINSIC CAUSES

Changes in the lung.—(*a*) *Collapse of the lung*, from whatever cause arising, is liable to be followed by bronchiectasis, and is one of the most common causes of that condition in children. In such cases the collapse is generally the result of capillary bronchitis. Stenosis and obstruction of a bronchus may, as we have already seen, lead to collapse of the corresponding area, and so favour the onset of bronchiectasis. Collapse of the lung secondary to pleurisy is considered under changes in the pleura (*vide* p. 131).

(*b*) *Pneumonia*, either lobar or lobular, may be an antecedent of bronchial dilatation, and either form of pneumonia, but especially the latter, may supervene upon the lesion already established. In a remarkable case lately under observation at the Middlesex Hospital, signs, which were clearly due to the dilatation of a bronchus at the apex, persisted after recovery from pneumonia of the left upper lobe, resolution having been incomplete. The patient had had seven attacks of pneumonia, for nearly all of which he had been an in-patient in the hospital, and, in all but the last, recovery of the lung had been complete. There was no sign of breaking down of the lung, and the absence of tubercle bacilli and elastic tissue from the sputa and the course of the attack negatived a diagnosis of tuberculosis. Without a knowledge of his history he would be now almost certainly regarded as a case of arrested tuberculosis of the apex. A pneumonia which has not undergone complete resolution may become in time a 'chronic pneumonia,' and with that condition bronchiectasis is very often associated.

Emphysema.—The same conditions which lead to emphysema may produce bronchiectasis, and a moderate degree of bronchial dilatation is not uncommonly observed in emphysema, but advanced emphysema and general bronchiectasis are rarely found in association.

Fibrosis of the lung arising from chronic pneumonia, tuberculosis, or other causes is frequently complicated by bronchiectasis, and various views are held as to the pathogenesis of the latter condition under such circumstances. Elaborate diagrams have failed to convince the writer of the correctness of the theory that traction by fibrous bands passing from the pleura to a bronchus or from one bronchus to another are the effective dilating force when bronchiectasis follows fibrosis of the lung. Moreover operations in cases of bronchiectasis have shown how commonly pleural adhesions, which are almost essential to the acceptance of this view, are absent when their presence has been regarded as practically certain.

Areas of fibrosis, whether of tubercular origin or not, tend to shrink by a process of centripetal contraction, and if the whole lung is affected the small space it may ultimately occupy is very striking. Chronic cavities tend to contract and their lumen may be almost (very rarely quite) obliterated; the parietal and visceral layers of the pleura become separated as the lung retracts, and the intervening space is filled by a fluid exudation which ultimately becomes organised into densely firm fibrous tissue, or the neighbouring tissue, if still pervious to air, becomes emphysematous. Organs are displaced and other changes occur, all of which appear to be more likely to obliterate than to enlarge spaces within the affected area. That they are not completely obliterated is due partly to the dilating force of the chronic cough, which is commonly present, and partly to the fact that when, by the aid of such changes as approximation and overlapping of the ribs, curvature of the spine, displacement of organs and of the mediastinum, and enlargement of the opposite lung, every possible compensation has been made, equilibrium between the various forces is obtained before the cavities in the lung have been completely obliterated.

Where a dilated bronchus is found opening into an irregularly shaped chronic tubercular cavity, neighbouring fibrous tracts may have had some share in producing the irregular outline, by preventing contraction rather than by acting as a dilating force; but this is not, according to our view, an agent of any importance in the production of bronchiectasis.

Changes in the pleura.—Pleurisy with effusion having led to collapse of the lung, either as a whole or in part, dilatation of the bronchi and chronic thickening of the interlobular and interalveolar connective tissue may follow. It is probable, as suggested by Wilson Fox, that these changes proceed together, and that the dilatation of the bronchi is not due to the fibrosis of the lung, but to the distending force of the cough acting upon the weakened tissue of the bronchial wall. The question discussed above arises in connection with these cases also, but it is unnecessary to recapitulate the arguments there advanced. Such fibrous tracts or bands as are not due to the presence of chronic interstitial pneumonia are in most cases the thickened interlobar or interlobular septa.

Inspiratory pressure.—The fact that the expansion of the affected part during inspiration is practically *nil* tells strongly against the effectiveness of that force as a factor in producing dilatation of the tubes.

Clinical varieties of bronchiectasis.—Two varieties of the affection are observed, the *acute* and *chronic*. The former, which is more commonly met with in children, may be produced in a comparatively short time as a result of whooping cough, diphtheria, acute and capillary bronchitis, broncho-pneumonia, pulmonary collapse, or pleurisy. (*Vide* Bronchiectasis in Children, p. 143.)

Cases of this type may occur in adults from similar causes and are not uncommon in typhoid fever. In a fatal case of that disease lately under the care of the writer cylindrical dilatation of the tubes was present in an area of consolidation produced by the coalescence of numerous foci of broncho-pneumonia; one of the dilatations nearly approached the surface of the lung This condition may lead to perforation of the pleura and pneumothorax.

Symptoms.—In a disease owning such a varied etiology as bronchiectasis uniformity in symptoms is not to be expected, but in well-marked cases they are usually fairly typical.

A paroxysmal *cough*, accompanied by the expectoration of a e quantity of purulent and often offensive secretion, is the combination of symptoms which most commonly suggests the presence of dilatation of the tubes.

In the intervals between the paroxysms the patient may be fairly free from cough, except when there is much emphysema, the cough is then usually more frequent and may be painful.

The dilated tubes lose their sensibility to a great extent and cough is not excited until, from the accumulation of secretion, healthy parts of the mucous membrane are irritated. Copious expectoration then follows.

The paroxysms tend to occur chiefly on lying down at night and rising in the morning, or when the patient assumes some particular position, generally the recumbent on the healthy side. The secretion then overflows into unaffected or less diseased bronchi and cough results.

The site of the affection materially influences the severity of the cough. When the lesion is chiefly at the apices or in the area of the upper lobes from which there is free drainage, there is not the same tendency to accumulation as when the lower lobes are mainly affected.

Expectoration may be easy, or effected with difficulty, owing to the presence of advanced emphysema or stenosis of a main bronchus or of marked induration of the tissue around the dilated tubes.

Characters of the sputa.—It may be some years before the sputum presents an offensive odour; then, for perhaps as long a period, a faint putrid smell is noticeable; subsequently when bronchiectasis is fully developed, the sputa, owing to putrefactive changes, acquire the horribly fœtid character which is such a marked clinical feature of the disease. The odour is not quite the same as that of gangrene; it has been compared to that of rotten cabbage; but a more precise description is hardly necessary, as it will be readily appreciated, possibly at a distance from the patient. It may be more marked in the breath than in the sputa or *vice versa*. We have known it to be so horrible that, if the patient coughed, vomiting was immediately induced in persons standing near him.

The expectoration is usually diffluent, seldom nummulated. After standing for about twenty-four hours it settles into either two, or more often three, well-defined strata—an upper thin, brownish, frothy mucoid layer, a middle layer of greenish fluid, either clear or containing hanging threads, and a thick lower layer of purulent material, which forms a grey opaque mass. Where only two layers are present the upper is more or less stringy, or it may be clear, the solid matters sinking to the bottom. In the latter, on microscopical examination, pus cells, granular detritus, connective tissue, and many organisms are found, amongst which micrococci, bacteria, spirals and filaments of leptothrix are the most numerous. Traube's or Dittrich's plugs, which are soft, fœtid, greyish-yellow bodies, formed in the bronchi and consisting of pus cells, granules, oil-globules and needle-shaped crystals, can usually be found in the lower layer by careful search. In some cases casts of the bronchi may also be found after floating a part of the deposit in water. Leucin and tyrosin may also be present.

The *quantity* of the expectoration varies, but is usually profuse and may amount to 25 or 30 ounces or more during the twenty-four hours. In a case recently under the care of the writer it averaged 163 ounces a week for a period of six months. The *reaction* may be alkaline, or it may become acid from the presence of butyric, lactic, valerianic and acetic acids.

Dyspnœa.—The breathing is not, as a rule, much quickened in the early stages, and then there may be no dyspnœa, but it is usually present to some degree on exertion, and it may be severe when cardiac complications exist. It is due rather to the co-existence of bronchitis, emphysema, or fibrosis than to dilatation of the tubes. During intercurrent attacks of bronchitis or pneumonia dyspnœa may be severe, as it almost invariably is towards the end of the case.

Hæmoptysis is usually a late symptom, but its occurrence is by no means uncommon. It was present at some period in 14 out of the 35 cases analysed, and in several it was profuse in quantity and frequently repeated. Only three of these were cases of tuberculosis, and in one there was aortic stenosis. An ulcerative process starting in the bronchial wall may extend to a neighbouring branch of the pulmonary artery and so give rise to hæmorrhage, or an aneurism on a branch of the pulmonary artery may project into a dilated tube, and the rupture of the sac may be followed by immediate death from hæmoptysis. In three of the cases hæmoptysis was the immediate cause of death, and in two of these a pulmonary aneurysm had ruptured into a dilated tube.

Pyrexia.—There may be no pyrexia throughout the case. In the early stages it is often absent, but if secretion is retained the temperature will rise; and towards the termination of a case, if septic broncho-pneumonia occurs, there is nearly always high fever of a remittent type. If death occurs from gradually increasing cyanosis, the temperature is generally sub-normal. Remitting attacks of fever are apparently due to retention of secretion, or to localised pneumonia.

Pain in the chest is usually due to pleurisy and is of importance, as possibly showing that the affection is approaching the surface of the lung. In cases originating in an attack of acute pleurisy there will almost certainly be a history of severe pain on the affected side.

Diarrhœa may result from septic infection, or may be due to lardaceous disease of the intestine.

Albuminuria is common from amyloid disease of the kidneys.

Clubbing of the fingers and toes, and also to a less degree of the nose, is such a marked feature of bronchiectasis as to be of decided value in diagnosis.

Course.—In cases originating in chronic bronchitis certain fairly well-defined stages of the affection may usually be recognised.

1. Expectoration is profuse and purulent, but not fœtid. Pyrexia is absent, strength and nutrition are well maintained. Exacerbations often occur in the winter months.
2. The expectoration acquires a faintly fœtid odour and becomes more profuse in quantity. This change may be accompanied by the signs marking the following stage, but it may precede their onset by a long period. There may be slight pyrexia, but it is rarely continuous, being, as a rule, limited to periods when the sputa are diminished in amount or entirely absent.
3. The condition is now fully developed, the sputa are decidedly fœtid, characteristic physical signs are present, and clubbing is well marked.
4. Pleural adhesions are formed as the result of an attack of acute pleurisy, or of one of a subacute type in which a 'dry' friction sound is present over a large area for a considerable period.
5. Fibrosis of the lung is followed by retraction and thickening of the pleura. Hæmoptysis may occur, and may prove fatal. Emaciation, dyspnœa, and possibly pyrexia are present.
6. Absorption of the septic contents of the tubes induces pyrexia of a remittent type, or possibly a rigor and severe general symptoms. Death may be due to pericarditis, septicæmia, pyæmia, cerebral abscess, nephritis, diffuse bronchitis, or septic broncho-pneumonia, with necrosis of the consolidated areas.

Cases originating in an attack of acute pleurisy or pneumonia may run a shorter course, *e.g.* a male, aet. 38, previously healthy, had pleurisy (L) followed in three weeks by cough and expectoration, which became copious and offensive in three days. Three severe attacks of hæmoptysis followed, and in six months from the onset death occurred from the rupture of a pulmonary aneurysm into a dilated bronchus.

In other cases the course of the affection is too variable to admit of analysis, but the three later stages described above may often be recognised.

Physical examination.—To describe in detail all the physical signs which may be met with in cases of bronchiectasis would serve no useful purpose, as the condition may be a secondary result of such a variety of diseases, and many of the signs pertain to the primary affection rather than to the bronchial dilatation.

The physical signs will necessarily depend upon the nature of the primary affection, the extent of the lesions, the condition of the surrounding lung, the amount of secretion in the dilated tubes, and upon their distance from the surface of the chest.

Inspection.—The chest may be markedly emphysematous in type, or with localised disease there may be retraction of the chest wall and displacement of the cardiac impulse.

In some cases, expansion, though only slightly impaired over the upper lobes, is decidedly diminished at one or both bases.

Palpation.—The vocal fremitus and resonance are increased in cases accompanied by fibrosis, and bronchophony or pectoriloquy may be present.

Percussion.—The note obtained will depend upon the extent of the lesion and the condition of the lung surrounding the dilated tubes. If the lung is healthy or emphysematous, the note will be either normal or hyper-resonant, but when the condition is well marked and numerous dilatations are present in a given area, the note is usually raised in pitch and deficient in resonance, but the limits of impairment are not, as a rule, sharply defined. Where a whole lobe is converted into a mass of saccules, the note is tympanitic, or, if the saccules are very large, amphoric. The tympanitic quality may be more decided and the pitch higher when the mouth is open.

Fibrosis around dilated tubes which are filled with secretion gives rise to an absolutely dull sound on percussion ; when they are emptied, the note is raised in pitch, and may present various degrees of dulness up to a distinctly wooden note.

Auscultation.—Cylindrical dilatations give rise to bronchial breath sounds of a blowing and rather hollow, but not cavernous quality. Both on inspiration and expiration the sound may be interrupted and wavy. With sacculated dilatations the breathing may be truly cavernous, and gurgling râles may be present. The 'veiled puff' of Skoda may often be heard. It is a curious sound occurring at the end of inspiration and giving the impression that a puff of air has suddenly entered a small cavity situated just beneath the ear. When once recognised it is easily distinguished, and is in our experience more often present in bronchiectasis than any other condition.

The character of the adventitious sounds depends to some degree upon the state of the surrounding lung. If this is spongy, they are 'bubbling;' if indurated from fibrosis, they become crackling in quality. The typical sound is a coarse râle of ringing or metallic quality. It may be almost constantly present over a certain area, or it may disappear and reappear according to the quantity of secretion in the tubes. A variety of other râles may be present, resulting from the co-existing bronchial and pulmonary lesions.

Râles are not infrequently produced in bronchiectasis by the movements of the heart, and a loud systolic murmur is in some cases audible about the cardiac apex, also posteriorly and in the trachea and mouth. In one very marked example of this kind under the care of the writer a systolic murmur could be heard at a distance of two feet from the patient when the mouth was open, d its presence had led to the suspicion of aneurysm.

Diagnosis.—The following sequence of events clearly and edly observed is almost diagnostic of bronchiectasis, viz. the expectoration of a large quantity of muco-purulent fluid, fœtid or not, followed by the discovery of cavernous breathing over a certain area, and the gradual disappearance of this sign as the secretion re-accumulates. The only other condition likely to be attended by similar signs is that which follows the rupture of an empyema into the lung. A case may be seen many times without this sign being observed, and it may, nevertheless, be present every day. The patient usually knows when the greatest amount of expectoration will be brought up, or he may be able, by assuming a certain position, to induce a free flow. If the examination is made before and after this occurs, the alteration in the signs over a certain area may in some cases be readily recognised.

The diagnosis from *chronic bronchitis with emphysema* is often difficult; it depends chiefly upon the character of the expectoration and the peculiar mode of its evacuation. Persistent fœtor points strongly to bronchiectasis, although in cases of 'putrid bronchitis' this condition of the sputa may be present, even though the tubes are not dilated. If the breath sounds are of the wavy bronchial quality, and are accompanied by coarse bubbling or ringing metallic râles, and particularly by the 'veiled puff,' it is very probable indeed that the tubes are dilated. When the signs are localised, and there is extreme fœtor, with evidence of breaking down of the lung, in addition to consolidation, the diagnosis lies between pulmonary gangrene and bronchiectasis followed by ulcerative and destructive changes. The former condition is generally a sequel of acute pneumonia, broncho-pneumonia, or tuberculosis; the latter is often the mode of termination in cases of simple bronchiectasis. The history and the results of examination of the sputum usually throw light upon the true nature of the case.

The diagnosis from *tuberculosis* rests mainly upon the results of the examination of the sputum, but it must be remembered that tuberculosis may be engrafted upon bronchiectasis, although such an event is not of common occurrence. The fact that the disease has not followed what may be called the ordinary 'line of march' of tuberculosis is a presumption against that diagnosis. Difficulty may, however, arise when a primary tubercular lesion in the upper lobe has undergone arrest, and in such a case the presence of the old lesion may be overlooked, and basic disease, although really secondary, may appear to be primary. Such cases are by no means infrequently met with.

Fortunately, however, an exact pathological diagnosis is then of comparatively little importance. It is otherwise, however, when the diagnosis lies between a *limited empyema* with an opening into the lung and chronic pneumonia with bronchiectatic cavities in the lower lobe, as in the former case an operation is decidedly indicated, whereas in the latter it is rarely advisable. We are not now considering those rare cases in which a single large bronchiectatic cavity is present in the lower lobe, as in such an operation is admissible and has been attended with success.

An *empyema* which has opened into the lung is a form of pneumothorax, and may be attended by the ordinary signs of that condition. In such a case the diagnosis is not as a rule difficult, but it is often almost impossible to distinguish between a loculated empyema situated between the lung and the diaphragm, or between the lobes of the lung with a perforation into the lung, and a cavity within its substance, and such a cavity may be bronchiectatic.

In such a case puncture with an aspirator needle may aid the diagnosis; but the proceeding is not without risk, as, if adhesions are absent, pus or air may enter the pleural cavity, or, as in a case reported by Dr. Carr, in which firm pleural adhesions were present, general subcutaneous emphysema may follow from the air being forced along the track of the needle during the act of coughing.

A case of pulmonary disease, marked by long-continued copious fœtid expectoration, from which tubercle bacilli and elastic fibres are absent, by diffuse bronchial rather than limited cavernous or amphoric breathing, by coarse bubbling or metallic rather than gurgling râles, and by extreme clubbing of the fingers, toes, and nose, is probably one of bronchiectasis, in which many tubes have undergone dilatation.

Prognosis.—When the affection is a sequel of measles or whooping cough in childhood, recovery, possibly complete, may follow a moderate degree of dilatation. Often, however, it becomes chronic; but even then it may for many years take the form of a severe bronchitis aggravated by inclement weather. As a rule, the course of the disease is very chronic, and cases are on record in which it had been present for so long a period as forty or fifty years. When it originates from an unresolved pneumonia or follows upon collapse of the lung, the result of pleurisy with effusion, recovery is extremely rare, and the course is not usually so prolonged as in cases secondary to chronic inflammation of the bronchi.

The chief dangers are from profuse broncho-pneumonia and gangrene, hæmoptysis, and cerebral abscess.

Cardiac and renal complications are common precursors of death.

Treatment.—To prevent the decomposition and to diminish the quantity of the bronchial secretion are the main objects of treatment. For these purposes inhalations of creasote, carbolic acid, tar, and turpentine have long been in use, and these and similar remedies have been administered internally. Such treatment cannot, however, be said to have been attended with marked success. This is doubtless due in great part to the fact that the quantity of the remedial agent which reaches the affected tube by the ordinary use of sprays and inhalations is insufficient to effect any decided change in the local conditions. Recently, however, other methods of treatment have been suggested, from which more decidedly beneficial results may be obtained. These are the continuous inhalation of the vapour of coal-tar creasote, or cresoline, the use of intra-tracheal injections, and of hypodermic injections of sterilised oil of guaiacol or of creasote.

right chest, but somewhat diminished towards the base. No tubercle bacilli were found in the sputum. The diagnosis was 'bronchiectasis of the right lower lobe with incomplete consolidation; dilated tubes in both upper lobes.'

Ten days after admission, the temperature assumed a markedly hectic type—99° in the morning and 103°–104° F. in the evening. For a month subsequently it was irregular, ranging between 99° and 101° M. and 102° and 103° E. The sputum during this period remained offensive, and averaged about 12 oz. daily. He was given 30 minims of sterilised oil of guaiacol by subcutaneous injections daily.

From May 30 to June 24 the fever continued, and the expectoration was exceedingly fœtid. On June 25 and 29 hæmoptysis occurred (2 oz. and 1 oz.). He continued to lose strength, and the râles at the right base became larger, and of metallic quality. Dulness appeared over right middle lobe. The sputum was still free from tubercle bacilli and elastic tissue. A very few streptococci were present, but no other organisms.

Towards the end of July the expectoration diminished to about 5½ oz. daily, and the fœtor was less. The temperature varied between 99°–101°.

During the first week of August he was not so well, the appetite fell off, pyrexia was more marked, and the sputa increased in quantity and became much more offensive.

From August 5 to 12 the patient had a creasote vapour bath for fifteen to twenty minutes on alternate days. The temperature had touched normal on August 4, but it remained so during the week. The sputum was slightly bloodstained, but not quite so offensive as previously. He gained 1 lb. in weight. The signs in the chest were unchanged.

A fortnight later his condition was much improved. The sputum was only very slightly offensive, and the temperature was normal.

During September the improvement continued. The quantity of expectoration was from 3 to 5 oz. daily. It was only slightly offensive, and often had no odour. There were very few râles audible, and the patient gained more than a stone in weight during the month.

The time spent in the bath was gradually increased, and during October was about an hour and a half daily.

On his discharge the following note was made:

'The patient looks a healthy man. He has improved to a considerable degree, having come in and remained for some time in an apparently hopeless condition. The improvement has been especially marked since the commencement of the creasote vapour bath treatment.'

Accurate notes of this case were taken by Dr. F. E. Leyton, house physician, to whose assiduous care the improvement in the condition of the patient was in great measure due. The writer cannot recal any case of chronic pulmonary disease in whi recovery followed from a condition apparently so hopeless, and the result may be credited to the treatment adopted. It is unfortunate that such a favourable result cannot always be looked for.

In a case recently under the treatment by creasote vapour baths for twenty-seven weeks, the weekly average of the sputa was almost exactly 100 oz., and during the last seven days the quantity expectorated was larger (116½ oz.) and more fœtid than for any other period. This patient took creasote internally during the whole of this period, and had a creasote or cresoline vapour bath for half an hour nearly every day, and for a few days he also had intra-tracheal injections of guaiacol.

In another case under treatment by baths and intra-tracheal injections (creasote ♏iv; sol. menthol (20 per cent.) ♏vi; ol. olivæ ad ℥j) for ten weeks, in which the sputum had been fœtid for five years, the odour disappeared in three weeks, but subsequently slight fœtor was present for a short time, but again disappeared. After the baths had been discontinued the expectoration remained free from any offensive smell. There was, however, no diminution in the quantity of the sputa.

The following is a brief account of the results obtained in a case of bronchiectasis of both lower lobes.

During 28 weeks 108 creasote and 26 cresoline vapour baths were taken, and 69 intra-tracheal injections of guaiacol were given. The quantity of sputum in the week before the treatment was 109 oz. Subsequently, in six periods of four weeks each, the average per week was as follows:

First month	160 oz. per week
Second „	175½ „
Third „	165 „
Fourth „	170 „
Fifth „	161 „
Sixth „	149 „

This shows that a considerable increase in the quantity of the sputa followed the use of the baths (not necessarily an unfavourable effect), but it subsequently continued profuse, although diminishing somewhat towards the latter part of the time. The fœtor disappeared after the treatment had been continued for seven weeks and did not return.

The gradual diminution, followed by complete absence of expectoration which may attend the use of this mode of treatment, is illustrated by the following case:

Edith T., æt. 14, admitted into the Brompton Hospital under the care of Dr. Fowler on May 14, 1897. Cough and copious expectoration had been present for eight years. Diagnosis—'Chronic endocarditis (mitral stenosis); sacculated bronchiectasis with much retraction of the left upper and lower lobes; com-
ncing bronchiectasis in the right lower lobe.' The sputum was
ly offensive. The patient took a creasote vapour bath for an
a half daily for six weeks. The total quantity of the

expectoration for six successive periods of one week each was as follows:

First week	45 oz.
Second ,,	43½ ,,
Third ,,	32 ,,
Fourth ,,	25 ,,
Fifth ,,	5 ,,
Sixth ,,	—

This patient gained 7 lbs. in weight, the cough disappeared, and the moist sounds present in the lungs on admission were no longer audible.

With our present experience of the creasote vapour bath treatment of bronchiectasis we have come to the following conclusions:

(1) It presents no practical difficulties and is unattended by any unfavourable results. The benefit to be obtained from the use of creasote vapour baths in bronchiectasis is far greater than from any other form of treatment.

(2) In exceptional cases a condition almost amounting to cure may follow their prolonged use.

(3) As a rule the fœtor of the sputa is modified, and not unfrequently the odour disappears altogether, but in some cases it is unaffected.

(4) The quantity of the sputa is not always or even as a rule diminished. It varies much from time to time and may be increased.

(5) The general condition of the patients improves, and those symptoms which remain cause less distress.

(6) The treatment requires to be continued for a long period, possibly as long as six months or more. In some cases its continuous use is advisable.

Remedies such as copaiba, santal oil, myrtol, eucalyptus, the balsams and gum resins, also creasote, tar and terebene, which tend to diminish excessive bronchial secretion, may be administered internally at the same time that the above treatment is being carried out; but we have not found this to be generally necessary.

Cod-liver oil, iron and quinine may be also given for their effect upon the general health.

A dry climate is indicated for cases in which profuse expectoration is present, and also one free from sudden changes of temperature, which are likely to induce fresh catarrh of the air passages. The affection is not, we think, common amongst well-to-do patients; but for such patients as can bear the expense the dry air of the desert would be most suitable, and they might pass the winter in Egypt. In this country Bournemouth can be recommended. Whilst the expectoration continues fœtid it is almost impossible for sufferers from this affection to mix with others in the ordinary life of an hotel, more particularly as they are often themselves unaware of the horrible odour which they exhale.

J. K. F.

CHAPTER IX

BRONCHIECTASIS IN CHILDREN

	PAGE
Etiology	143
Morbid anatomy	143
Symptoms	147
Diagnosis	148
Prognosis	148
Treatment	149

DILATATION of the bronchi is not uncommonly met with in children, and in some cases the affection does not present any features which differ essentially from those observed in adults; in others it runs a very acute course, and the finest ramifications of the tubes undergo dilatation, a condition to which the term 'bronchiolectasis' has been applied. As pointed out by Dr. Walter Carr,[1] to whose interesting paper the writer is much indebted, the subject has hitherto hardly received the attention which it deserves; this fact and the existence of the special features above mentioned render it desirable that a separate description should be given of the disease as it appears in young children.

Etiology.—The affection is most frequently observed in badly nourished syphilitic or rickety children who are attacked with bronchitis, which may be either primary or secondary to some acute specific disease, such as measles, whooping cough, or diphtheria. Such children are often the subjects of some deformity of the thorax, the result of previous disease.

Morbid anatomy.—The lesions which specially characterise the most acute cases are: (*a*) acute peribronchitis; (*b*) dilatation of the bronchioles throughout extensive areas of the lungs, or almost the whole of both organs; (*c*) the presence of innumerable small cavities, which give the lungs a worm-eaten or honeycomb arance; and (*d*) the presence on the surface of the lungs of l vesicles containing air.

---

onchiectasis in Young Children,' *Practitioner*, Feb. 1891.

These changes may occur in association with miliary tuberculosis of the lungs, but in the majority of cases hitherto recorded tubercle has been absent.

In chronic cases the changes are such as have already been described.

The following were the appearances in a case of acute bronchiectasis recorded by Dr. Sharkey.[1]

'The lungs were pale and curiously dotted with black pigmented spots. These were hard to the touch, and the centre of each was occupied by a small bronchus. Microscopically an acute peribronchitis was found accompanied by extreme bronchiectasis, a certain amount of acute interstitial pneumonia, apparently spreading from the acute peribronchial inflammation and a little, but very little, emphysema. The walls of most of the finer bronchi were infiltrated with leucocytes, which invaded the peribronchial connective tissue in large numbers, forming a solid circular mass which was perforated by the dilated lumen of the bronchus. From these as centres the leucocytes invaded the walls of the surrounding alveoli, but those at a distance from the bronchi were but little altered; catarrhal pneumonia was conspicuously absent. No tubercles were seen. The patient was a child aged two years. Death occurred twelve days after the onset of diphtheria. The child had previously enjoyed good health, with the exception that she had suffered from an attack of measles.'

In another case of bronchiectasis of a rather less acute type, also reported by Dr. Sharkey,[2] the following condition was found post-mortem:

'The lungs were very bulky; their surfaces were thickly strewn with small, round, transparent, bladder-like elevations. On section both lungs showed a precisely similar condition. A number of small cavities, the largest of about the size of a pea, were scattered through the organs and gave them a worm-eaten appearance. The cavities had perfectly smooth walls, and were either empty or full of frothy mucus. They were more thickly distributed under the pleural surfaces.

'The larger bronchi were not perceptibly dilated and showed no sign of disease. In both lungs there were numerous patches of broncho-pneumonia, all of small size, and here and there was some collapse. There were no signs of tubercle. The bronchial glands were healthy. Microscopically it was seen that there was widespread but unequally distributed acute bronchitis with peribronchitis, interstitial and alveolar broncho-pneumonia and pulmonary collapse. The bronchioles were extremely dilated and their walls thinned, and there was in addition considerable emphysema.'

The patient was a child aged four years, who had previously enjoyed good health. He had suffered from cough for two months, accompanied by expectoration of thick phlegm and vomiting, the latter occurring three or four times a day. On admission the child was poorly nourished, presented a dusky flushed face, and was breathing very rapidly (R. 44). Cyanosis developed, and crepitations became audible over the whole of both lungs. About a week before death, which occurred ten weeks after the onset of the illness, subcutaneous emphysema of the head, neck, arms, thorax and abdomen appeared, and post-mortem mediastinal and interstitial emphysema was found.

---

[1] *St. Thomas's Hospital Reports*, vol. xxii.

[2] *Loc. cit.*

In a similar case, occurring in a boy æt. 18 months, reported by Dr. Howard Tooth,[1] under the heading 'Multiple Cavities in Broncho-pneumonia,' the lungs were thickly and uniformly studded with minute smooth-walled cavities from $\frac{1}{16}$ to $\frac{1}{8}$ of an inch in diameter. The cavities contained air only, and no direct communication between them and the bronchi could be discovered. On microscopical examination there was evidence of broncho-pneumonia and acute peribronchitis. The walls of the cavities were for the most part formed by masses of inflammatory cells, but in some traces of an elastic lamina were observed. These cavities were believed to be dilated bronchioles, the dilatation being the result of the peribronchial inflammation.

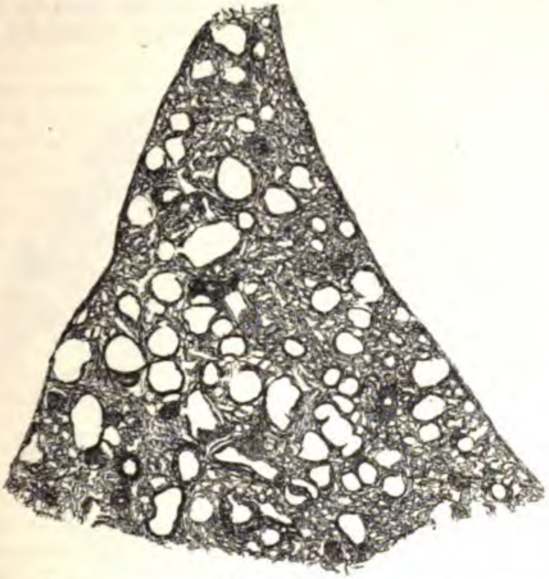


FIG. 58.—SECTION OF A HONEYCOMB LUNG (× 7 diameters)

In a case recently observed at the Middlesex Hospital, similar lesions were present in association with tubercle of the lungs, thus ming the opinion expressed by Dr. Voelcker in the discussion followed the reading of Dr. Tooth's paper that there are two varieties of this form of acute bronchiectasis, the one tubercular and the other non-tubercular.

---

[1] *Path. Soc. Trans.* 1897, p. 30.

The naked eye appearances of the surface and of a section of the lung in this case were precisely similar to those described by Dr. Sharkey in the second of his cases, but the microscopical appearances were somewhat different, as numerous miliary tubercles were present throughout the lungs.

Fig. 58 is from a section of a portion of the lung from this case, magnified seven diameters. It shows clearly that the bladder-like elevations on the surface are similar to the minute cavities within the substance of the lung. They have been mistaken for the beads of air seen on the surface of the lung in cases of interstitial and subpleural emphysema, but their distribution is quite different, as they do not occur in rows, and also they are not movable on pressure.

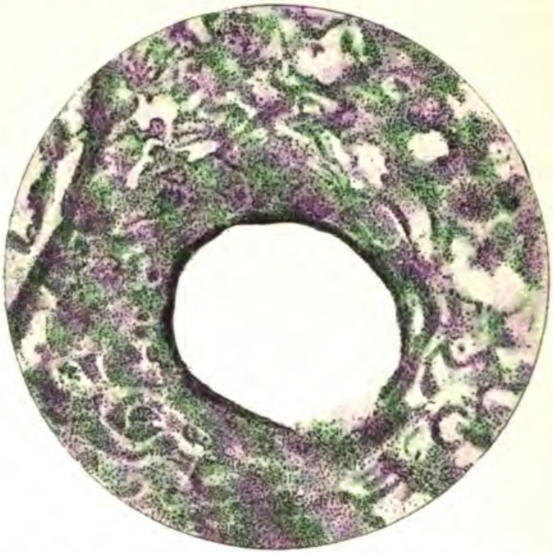


FIG. 59.—SECTION THROUGH A MINUTE CAVITY IN A CASE OF HONEYCOMB LUNG (low power)

The microscopical appearances are shown in fig. 59. The walls of the cavity are seen to be formed by masses of inflammatory cells.

The exact pathology of the condition known as 'honeycomb lung' is not yet determined. By some observers it is doubted whether, at any rate in the tubercular cases, the cylindrical dilatations are really dilated bronchioles, and it is certainly rarely possible to demonstrate the presence of any structure belonging to the bronchiole in the cellular exudation forming the walls of the dilatations. In some cases the larger cavities appear to be formed by the breaking down of septa intervening between neighbouring spaces.

After the above was in type the report appeared of Drs. A. A. Kanthack and H. D. Rolleston,[1] to whom Dr. Tooth's specimen (*vide supra*) was referred for examination. It is as follows: 'While agreeing generally with Dr. Tooth's description we would point out that the alveoli are almost universally altered, and show the effects of inflammation of some standing. Their walls are thickened and infiltrated, and the partitions are frequently broken through, so that several air spaces are converted into one large cavity. In many places groups of alveoli are obliterated by round-cell infiltration, especially around the bronchioles; in other places alveoli are obliterated by tissue showing the stages of organisation, and in these situations giant cells may be seen sometimes in considerable numbers. The bullæ are vomicæ formed by the breaking down of broncho-pneumonic masses, and are not bronchiolectases. The process being primarily a broncho-pneumonia, breaking down of scattered consolidated areas has led to the formation of abscess cavities. These vomicæ can in some places be seen to open into the bronchioles. Although there is no definite evidence of tubercle we do not in the absence of inoculation experiments consider that it has been excluded.'

In the acute cases the right cavities of the heart are, as a rule, found to be greatly dilated after death.

In cases which are not of the acute type hitherto described either saccular or fusiform dilatations may be present. The former are most common when a general bronchitis or peribronchitis is the cause of the affection, whilst cylindrical or fusiform dilatations may occur in areas of broncho-pneumonia, or from localised lesions of the bronchi, or from the presence therein of foreign bodies. In cases of long standing, chronic pneumonic changes may occur in the lung tissue surrounding the dilated tubes and lead to fibrosis and induration followed by retraction of the lung. In such cases firm pleural adhesions are usually found post-mortem.

Symptoms.—The child is generally anæmic and emaciated and suffers from cough, which tends, as the lesion advances, to be paroxysmal and may closely simulate whooping cough. The breath will probably not be fœtid and expectoration may be absent, because it is swallowed; but during the act of vomiting, which may accompany the cough, large quantities of pus may be expelled. Fever is not necessarily present, but during some period of the disease a high temperature (103°–105°) is generally observed. The pyrexia may be of short duration only, and may be clearly traceable to an intercurrent attack of pneumonia, which may be followed by an extension of the area of the disease. Towards the close, when cyanosis is present, the temperature is generally subnormal. In subacute and chronic cases clubbing of the fingers is often observed. Notwithstanding the presence of somewhat severe symptoms, the general condition of the patient may remain fairly good, and may be much less unfavourable than would be expected from the extent of the disease within the lungs.

---

[1] *Path. Soc. Trans.* 1897.

The **physical signs** do not differ materially from those already described. The *percussion* note may be normal, or it may be high pitched and deficient in resonance. With localised disease, especially of the lower lobes, there may be decided dulness. The *breath sounds* may be tubular or hollow or cavernous over the site of disease, and accompanied by sharp clicking or ringing râles, which, as the dilatations gradually increase, become gurgling and cavernous in character. In acute cases, however, tubular breathing may be absent, and crepitant râles may be audible over extensive areas. Other adventitious sounds may be present, due to coexistent bronchitis or pneumonia, *e.g.* sibilant and sonorous rhonchi, small crackling, or fine or coarse bubbling râles. The special feature of the adventitious sounds is their variability from day to day both in number and quality.

Diagnosis.—The absence of expectoration often renders the diagnosis between bronchiectasis and tuberculosis more difficult in children than in adults, and from the physical signs alone it may be almost impossible to distinguish the two conditions, and, as already stated, acute bronchiectasis and tuberculosis may coexist; but marked variability in the signs present from day to day is characteristic of bronchial dilatation. There is often in bronchiectasis a decided want of proportion between the extent of the disease, as evidenced by the results of the examination of the chest, and the general condition of the patient. The latter may indeed improve whilst the disease is extending—a condition rarely observed in tuberculosis. The moderate degree of pyrexia or the absence of fever may also negative the presence of an acute tuberculous lesion.

The presence of glandular enlargement elsewhere is in favour of a diagnosis of tubercle. When bronchial dilatation is associated with tubercular lesions it may be impossible to determine the exact condition present.

Prognosis.—There can be no doubt that when rapid dilatation of the bronchi occurs as a result of the pulmonary complication present in certain acute diseases, recovery from the attack may be followed by contraction of the tubes, which may return to their normal condition; but such a favourable termination is hardly to be expected in weak and badly nourished children. Very often the condition passes into the chronic stage; but even then, although permanent, it is not necessarily progressive provided the patient is placed under the most favourable conditions.

Dr. Carr[1] very rightly insists, as we have done elsewhere in connection with pulmonary tuberculosis, on the necessity of taking all the features of the case into account in forming a prognosis, viz. the history, general condition, symptoms, degree of pyrexia and the physical signs. If attention is directed chiefly to the latter a too unfavourable opinion is almost sure to be formed. In the most acute cases in which the condition occurs as a complication of diphtheria, death may take place in as short a period as twelve days;

but the fatal termination is then probably due chiefly to the influence of the primary disease. In some cases in young children the disease has apparently lasted for three or four years or more, and in older children it may be of much more prolonged duration.

Treatment.—The most important indications are to shield the patient from anything likely to give rise to fresh catarrh of the air passages, to promote expectoration, and to maintain and improve the strength and nutrition, by giving as much food as can be assimilated, with cod-liver oil and tonic remedies. Inhalations of oxygen are indicated when there are signs of deficient aeration of the blood.

We have had no experience of the use of creasote vapour baths in bronchiectasis occurring in very young children. It is possible that they might be greatly alarmed by the necessary proceedings, but in older children (æt. 6 years and upwards) we have found this method of treatment of as much value as in adults.

Bronchiectasis is a comparatively rare affection amongst the children of those in affluent circumstances, and it is rarely practicable, even when it might be advisable, to send children abroad; but, if possible, the patient should be removed to a dry and sheltered place, such as Bournemouth.

J. K. F.

CHAPTER X

BRONCHIAL STENOSIS AND OBSTRUCTION

	PAGE
Symptoms	150
Physical signs	151
Diagnosis	151
Prognosis	151
Treatment	151

NARROWING and complete obstruction of a bronchus may arise from a great variety of conditions having their origin either without the tube, or in its structures, or within its lumen.

Incidental references to the subject of bronchial stenosis and obstruction and their effects upon the lungs will be found in the chapters on Mediastinal Tumours (p. 680), Diseases of the Bronchial Glands (p. 153), Stenosis of the Trachea (p. 77), Pulmonary Syphilis (p. 429), Bronchiectasis (p. 129), Foreign Bodies in the Bronchi (p. 426), and on the various forms of Bronchitis. As the condition is not of sufficient importance from a practical point of view to justify a recapitulation under this heading of all that has been said elsewhere, we shall limit our consideration to those features which are common to all forms of the affection.

Narrowing or obstruction of the smaller bronchi, which occurs in the various forms of bronchitis, in asthma and emphysema, need not detain us, as the clinical use of the term 'bronchial stenosis' is limited to cases of narrowing of a main bronchus or of one of the larger tubes. As stated under Bronchiectasis, gradual narrowing of a bronchus tends to produce accumulation of secretion and dilatation of the tubes beyond the point of constriction, and subsequently fibrosis of the surrounding lung, whereas sudden and complete obstruction leads to collapse of the area of lung to which the bronchus belongs.

The **symptoms** of stenosis of a main bronchus are dyspnœa directly proportionate to the degree of narrowing, and inversely to the compensatory changes in the opposite lung. The dyspnœa is often paroxysmal in character, especially in cases of compression from aneurysm. Cough and expectoration are usually present, and may have the characters always associated with bronchiectasis.

The **physical signs** of a moderate degree of stenosis of a main bronchus are diminished expansion of the affected side, recession of the supra-clavicular and episternal regions, of the costal margin and of the interspaces, with feeble breath sounds and diminished vocal fremitus and resonance over the affected side. Stridor may be heard in the interscapular region, and sonorous rhonchi may be produced at the site of constriction, and rhonchal fremitus may be felt.

When the condition has lasted some time, or the degree of narrowing is very considerable, the affected side may undergo retraction. Signs of bronchiectasis may be present, and there may be dulness on percussion over the affected side due to collapse, condensation, or fibrosis of the lung and thickening of the pleura. The breath sounds tend to become weaker as the lumen of the tube narrows, and are absent when the bronchus is completely occluded.

Diagnosis.—To determine the existence of narrowing of a main bronchus is not as a rule difficult, but to arrive at a pathological diagnosis is often extremely so. When doubt remains after a careful physical examination, the probabilities are in favour of compression by an aneurysm, or a mediastinal growth, or constriction from cicatrisation of a syphilitic gumma, in the order named. Feeble breathing over the left upper lobe, associated with the sign known as 'tracheal tugging,' points strongly to an aneurysm. In cases of stenosis from syphilis, the lower end of the trachea is generally also involved, and the signs of tracheal stenosis may mask those of narrowing of one of the main bronchi.

Long-continued pressure of a growth accompanied, as is generally the case, by infiltration of the wall of the bronchus, generally leads to bronchiectasis; but the same may be said of syphilitic stenosis.

There are cases in which a pathological diagnosis is almost impossible, as, for example, one long ago under the care of the writer, in which a chondroma growing from one of the bronchial cartilages had occluded the lumen of the tube.

The conditions which we have most frequently found to be overlooked are stenosis of a main bronchus from contraction of a syphilitic gumma, and obstruction from the presence of a foreign body. This is due to the fact that in cases of visceral syphilis the external manifestations of the disease have often been but slightly marked, and that when a foreign body is lodged in a bronchus, there may be nothing in the history of the onset of the illness obtained from the patient, who is not uncommonly a schoolboy, suggestive of such a diagnosis.

The **prognosis** in all cases of obstruction of a main bronchus is necessarily grave.

Treatment.—In cases of stenosis from syphilis and compression by an aneurysm, large doses of iodide of potassium are indicated, and this drug is worthy of a trial where an exact pathological diagnosis cannot be made. We have known paroxysmal dyspnœa due to the presence of an aneurysm much relieved by inhalations of chloroform, and also of oxygen. Hypodermic injections of morphia, atropine, and strychnine may also be given in such cases. The administration of ether and ammonia may afford relief by temporarily freeing the tubes from accumulations of mucus.

Tracheotomy has often been performed in such cases from an erroneous diagnosis of the site of the obstruction.

J. K. F.

CHAPTER XI

DISEASES OF THE BRONCHIAL GLANDS

PAGE
Etiology and morbid anatomy 153
  In acute diseases 154
    „ acute bronchitis 154
    „ chronic bronchitis 154
    „ broncho-pneumonia 154
    „ pneumonia 154
    „ pulmonary gangrene 154
    „ diseases due to the inhalation of dust 154
    „ mediastinal growths 154
    „ lymphadenoma 154
    „ syphilis 154
    „ abdominal and retro-peritoneal disease 154
  Tubercular diseases of bronchial glands 154
    Morbid anatomy 155
    General symptoms 155
Physical signs 156
Diagnosis 156
Prognosis 157
Treatment 157

THE lower tracheal and bronchial glands lie chiefly in the space formed by the bifurcation of the trachea and around the main bronchi. They vary from twelve to fifteen or more in number, the largest being situated below the bifurcation. Smaller glands accompany the bronchi within the lungs, where they lie in the interlobular connective tissue. They receive the subpleural, perivascular, and peribronchial lymphatics, and are in communication above with the tracheal and œsophageal glands.

Etiology and morbid anatomy.—The bronchial glands undergo enlargement in a great variety of diseases, and, independently of disease, are usually found to be of a deep black colour, from absorption of carbon or other pigment, in individuals who have lived in towns and have attained adult age.

It will suffice here to recapitulate briefly the conditions present in various affections in which the changes in the glands are unattended by obvious signs, a more detailed description being reserved for such as may be clinically recognised.

In *acute diseases*, such as measles, scarlet fever, and typhoid fever, but particularly in whooping cough, they may undergo enlargement.

In *acute bronchitis* the glands are swollen and hyperæmic, presenting an opaque appearance on section, and exuding a turbid fluid on pressure.

In *chronic bronchitis* they are often deeply pigmented and indurated. If death has been due to an intercurrent acute attack they may be swollen.

In *broncho-pneumonia* in children they are generally much enlarged and present an appearance sometimes termed 'medullary.' If the swelling is very considerable they may be pale; if it is not so great they present an appearance of hyperæmia.

In *pneumonia* they are much enlarged and may present a similar appearance.

Gangrene of the lungs is often accompanied by gangrene of the bronchial glands and gangrenous material may be present in the tributary lymphatics.

In *disease due to the inhalation of dust* they are often enlarged and always pigmented. In miners they are of a coal-black colour, in ironworkers of a reddish tint, in potters they may be black and pulpy and contain solid blackish grains.

In connection with *mediastinal growths* these glands may be enormously enlarged, compressing and occluding the bronchi. Lymphosarcoma may originate in them, as may probably other forms of new growth, and they may be secondarily involved in *malignant disease* of various parts of the body.

Lymphadenoma may and generally does involve the bronchial glands, which may form large masses and cause some degree of narrowing of the main bronchi in children. The glands present the ordinary appearances of that disease. They do not caseate or soften.

These glands may also be found enlarged in secondary and tertiary *syphilis*.

Disease originating in the *abdomen* or *retro-peritoneal tissues*, whether malignant or tubercular, may infect the bronchial glands, and subsequently those in the supra-clavicular fossæ on either side of the spine. Symmetrically enlarged glands in that situation should always suggest the presence of primary disease in the abdomen or posterior mediastinum, when lymphadenoma can be excluded and there is no lesion of the cervical glands.

TUBERCULAR DISEASE OF THE BRONCHIAL AND LOWER TRACHEAL GLANDS

The direction of the lymph stream being from the lungs towards the bronchial glands, it is natural that pulmonary tuberculosis should be the precursor of the affection in the great majority of cases. In children, however, the lymphatic glands share with the bones the pre-eminent position as regards tubercular disease, which in adults is occupied by the lungs, and in them it is by no means uncommon to meet with cases of very extensive, sometimes almost universal, tubercular infection of the glands, whilst the lungs show few or no signs of that disease.

In many of such cases the tubercle bacillus enters the body through the intestinal mucous membrane, milk from a cow with tubercular disease of the udder being the vehicle of the organism, In others it is absorbed from enlarged tonsils, passing from the cervical to the tracheal and bronchial glands. In others, but probably seldom in children, the organism is inhaled and primarily affects the lungs.

Morbid anatomy in tubercular disease.—Masses of enlarged glands are found along the trachea, at its bifurcation and around the main bronchi, and in smaller number they may be traced along the larger tubes. The appearances they present are various; in the most acute cases those last infected are inflamed, swollen, soft, and of a pinkish tint; at a later stage they are still swollen, but of a greyish colour, or they may be studded with miliary tubercles and present areas of caseation. The greater number of the glands, however, and sometimes all of them are caseous or are undergoing softening.

The presence of calcareous nodules and of a thickened fibrous capsule are signs of a long-standing lesion.

General Symptoms of Tubercular Enlargement of the Bronchial Glands

The symptoms of this affection vary in severity according to the degree of enlargement, being in some cases so slight as to escape recognition, and in others so obvious as to render the case clinically indistinguishable from one of mediastinal tumour.

In well-marked cases cough is usually present, and may be paroxysmal and of a character suggesting a laryngeal origin. It may, however, be short, frequent, and hacking. A spasmodic cough, suggestive of whooping cough, has been observed in a considerable number of cases. Pressure upon the pneumogastric nerve or both recurrent laryngeal nerves by enlarged bronchial glands may give rise to spasm of the adductor muscles of the larynx or to paralysis of the abductor muscles; and such conditions may account for the spasmodic cough and dyspnœa. Hoarseness or aphonia may also occur under these circumstances. Cough and dyspnœa may also arise from direct pressure upon the lower end of the trachea and the main bronchi. Acceleration or slowing of the action of the heart may also result from pressure upon the pneumogastric nerve.

Expectoration of a mucoid character is often present, or it may be purulent and contain tubercle bacilli when a softened gland has perforated a bronchus.

Hæmoptysis is not an uncommon symptom, but as a rule the quantity of blood is small.

CHAPTER XII

EMPHYSEMA OF THE LUNGS

PAGE

Pathogeny 159
- Theories of emphysema 159

Etiology 162
- Minute anatomy of the lung 163

Morbid anatomy 165

Varieties of emphysema 165
- Large-lunged 165
- Small-lunged 166
- Local 167
- Acute vesicular emphysema 167

Associated lesions 168

Large-lunged emphysema
- Symptoms 169
- Physical examination 171

Small-lunged emphysema
- Symptoms 173
- Physical examination 173
- Symptoms of local emphysema 174
- Symptoms of acute vesicular emphysema 174

Diagnosis 174

Prognosis 176

Treatment 176

Definition.—A disease of the lungs characterised by over-distension of the alveoli and atrophy of the alveolar walls.

It has been the custom to describe under this heading two essentially distinct morbid conditions; the one, corresponding in anatomical details to the definition above given, having nothing in common with the other but the name. An account of this latter affection, *interlobular or interstitial emphysema*, will be found in the chapter following.

The description of emphysema of the lungs given by Laennec, accurate though it was as regards both anatomical characters and clinical history, remained incomplete until supplemented by the microscopical researches of Rokitansky and the clear exposition of its pathology which we owe to Sir William Jenner. Our knowledge of the disease has been mainly derived from their writings, and few additions of importance have been made to it in recent years.

Pathogeny.—Various theories have been advanced as to the mode of origin of emphysema, some of which meet with but little support at the present time. It would serve no useful purpose to enter upon a detailed discussion of the question, as it is exhaustively dealt with in the original papers of Sir William Jenner, to which reference may be made. It will be sufficient to mention those views which have at any time received considerable support, and to discuss in greater detail that which is now generally adopted.

Primary degeneration theory.—The view that the general cause of emphysema is a primary fatty degeneration of the alveolar walls was first stated by Rainey, and subsequently received support from Villemin. The latter writer describes the changes as beginning in an excessive proliferation of the intercapillary nuclei, followed by secondary fatty degeneration of the nuclei and other structures, the result of pressure upon the capillaries. It is now generally considered that the degenerative changes in the alveolar walls are secondary to the distension of the air vesicles and interalveolar spaces, and to the diminution in the blood supply thereby induced.

It is possible, however, that in the form of emphysema met with in old people, primary degenerative changes may play a more important part. Reference will be made to this point subsequently.

Inspiratory theory.—The theory that emphysema is due to distension of the lungs during inspiration was really first advanced by Laennec. He believed that the air drawn into the lung in inspiration was retained, being unable to escape during expiration owing to the obstruction caused either by catarrhal swelling of the mucous membrane of the bronchi or by accumulation of mucus in the tubes, and that as a consequence the lungs became over-distended with air.

Gairdner in 1850 stated the inspiratory theory in a different form. According to his view some change in the lungs, such as collapse or retrocedent tubercle, leading to a diminution in size in one part, preceded the development of emphysema. As the air vesicles within the area of disease or collapse did not expand during inspiration, an undue strain was thrown upon those in the immediate neighbourhood by the incoming air, and in consequence they became enlarged.

This theory, as regards the general disease, has been completely displaced by that to be next mentioned; and as an explanation of the conditions found around patches of collapse or of fibroid tubercle—compensatory emphysema—it is believed that the distending force of inspiration, although possibly not without effect, is subordinate to that of forced expiration.

Expiratory theory.—In 1845 Mendelssohn first advanced the theory that emphysema is produced during a forced expiration. He believed that the air was prevented from escaping from the upper lobes by the pressure of the diaphragm during forced expiration; that consequently the tension within the lung was increased, and the air vesicles underwent dilatation. In 1857 Sir William Jenner stated the above-named theory in the following terms: 'The lung during expiration is compressed at different parts with different degrees of force. The parietes of the thorax, in consequence of their anatomical constitution, yield to the same force at different parts with various degrees of facility. The chosen seats of emphysema are exactly those parts of the lung which are the least compressed during expiration, and which are situated under those portions of the thoracic parietes that give way the most readily before pressure.'

In a footnote to his paper on 'Emphysema of the Lungs,' in Reynolds's 'System of Medicine,'[1] Sir William Jenner stated that he was unacquainted with Mendelssohn's paper when he advanced this theory in 1857; and that so far as he was aware the existence of that paper was unknown in this country until 1867, and rarely, if ever, referred to abroad until that date.

Having regard to the above facts, to the singular completeness of Jenner's papers, and to his demonstration of the exact sites of emphysema, we may fairly regard him as having been the first to make known the true mode of origin of the disease.

The increased tension in the air passages, which we have seen to be a common antecedent of emphysema, may be induced in various ways.

Cough.—The almost invariable association of some degree of emphysema with chronic bronchitis points to cough as the most frequent cause of the disease. The chest having first been filled with air, the glottis is closed, a violent expiratory effort is made during which the pressure within the air passages is enormously increased, the glottis then relaxes, the air passes rapidly through the narrow orifice, and a cough results. It is the frequent repetition of this act which eventually induces a permanent dilatation of the air vesicles and interalveolar passages. The effect of the compression of the lungs by the vaulted arch of the diaphragm during a violent expiratory effort, such as that above described, is to drive the air in all directions from the central to the peripheral part of the lungs; the result is the distension of those parts which are least supported. As pointed out by Sir William Jenner, these parts are the apices, the anterior margin of the upper lobes, and the margins of the bases of the lungs. These are the sites of the primary lesions; but in the course of the enlargement of the thorax which they entail, the relative position of a given area of lung and the chest wall gradually changes, fresh portions being brought into contact with the intercostal spaces, the resisting power of which is less than that of the ribs, and thus in course of time the change may become general throughout the lungs.

Muscular effort.—It is probable that next to cough violent muscular effort is the most common cause of emphysema. The mechanism is as follows: the lungs having been completely expanded by a deep inspiration the glottis is closed; any severe and sustained muscular effort with the thorax in this condition necessarily subjects the lungs to strong compression, the increase in pressure within the air passages being most effectual in distending the lung in those situations where the organ meets with least support.

Further reference will be made to causes of over-distension in describing the etiological factors of the disease.

It will be convenient here to refer to those conditions of a temporary nature which lead to over-distension of the air vesicles. In such cases when the cause is removed the effect may disappear, but whether it does so or not depends upon the duration of the exciting cause and the integrity of the elastic tissue of the lung.

The best example which can be given of this temporary over-distension of the lungs is the condition observed during a paroxysm of asthma. At the height of the attack the lungs may be found distended with air to a degree equal to that present in the most advanced cases of emphysema; but when the attack has passed off, the organs may return to their previous size. It is rare, however, to meet with patients whose asthma is of long standing who are not also the subjects of emphysema.

The mechanism by which this state of over-distension is produced appears to be a matter of doubt, the explanations given varying with the theory adopted as to the cause of the asthmatic paroxysm. If the theory either of a spasm of the diaphragm or of the muscles of inspiration is held, there is little difficulty in understanding why the chest is in a condition of extreme inspiratory distension; if, on the other hand, we reject both these views and accept that now generally received, namely, that the asthmatic paroxysm is due to bronchial obstruction, the result either of a spasm of the muscular fibres of the bronchi or of a fluxionary hyperæmia of the bronchial mucous membrane, the explanation of its mode of occurrence is not quite so obvious.

It is, as a rule, gradual in onset and also in decline, and is apparently brought about in the following manner:

(i) The bronchial obstruction induces increased inspiratory effort.

(ii) The entering air passes the obstruction with difficulty, but the gradually increasing prolongation and force of the expiratory act shows that the air meets with still greater difficulty in escaping from the lungs.

(iii) Expiration, although prolonged, is not sufficiently so to produce an equilibrium between the incoming and outgoing air; a fractional addition is therefore made to the residual air by each completed act of respiration, and in time the lungs become over-distended.

It may be objected that, as the force of expiration is greater than that of inspiration, the obstruction should be more easily overcome by the outgoing than by the incoming current of air; but it would appear that experience teaches every individual to rely upon forced inspiratory efforts to remedy a defective aeration of the blood, whereas the condition really requires for its relief forced efforts limited to the period of expiration.

Another possible factor in the production of this state of extreme distension is the compression of the smaller bronchi by the distended alveoli, an effect necessarily more felt during expiration.

Other causes of temporary over-distension of the lungs are laryngeal obstruction, from whatever cause arising, whooping cough, acute bronchitis in children, and severe muscular strain.

Etiology.—*Age.*—It is a matter of common experience that the disease may be met with at any age. Some of the most marked examples are seen in young children. The atrophic form of the affection (see under Varieties of Emphysema) is most often met with in old people.

Sex.—Men are naturally more subject to the disease than women, as they are more exposed to the conditions which favour its development.

Occupation.—Any occupation involving severe muscular effort, especially if performed with the lungs distended and the glottis closed, tends to produce emphysema. In all such efforts the chest is forcibly compressed by muscular contraction, and the act is equivalent to one of forced expiration. The classical example of an occupation involving the latter condition is that of a cornet-player. Smiths, hammermen, and porters engaged in lifting heavy weights are all liable to develop emphysema. Omnibus and cab drivers, and all whose occupations involve exposure to inclement weather, are prone to attacks of bronchitis, whence comes emphysema. The inhalation of dust, a condition almost inseparable from many occupations, necessarily induces catarrh of the bronchi; upon this cough and emphysema follow.

Diseases such as whooping cough and chronic bronchitis present the conditions essential to the production of emphysema to the fullest extent. The violent respiratory acts in many forms of dyspnœa may lead to extreme over-distension of the lungs, which may be either temporary or permanent. The same is true in cases of extensive collapse of the lungs as regards those parts into which the air is free to enter. The mode of production of emphysema in asthma and allied conditions has been considered above.

The onset of emphysema will naturally be favoured by any conditions, such as chronic congestion from valvular disease and chronic bronchitis, which tend to diminish the natural elasticity of the lungs. Advancing age is a factor which operates in a similar manner.

Hereditary predisposition.—It has been suggested that there exists in some individuals and families an hereditary tendency to the disease, but this view is rarely insisted upon at the present time. Various observers have investigated this matter, the result being the supposed discovery of the hereditary tendency in a proportion of cases varying from 12 per cent. (Lebert) to about 60 per cent. (Fuller, Jackson) in adults, and 100 per cent. of cases in children (Jackson). It is probably true, as pointed out by Sir William Jenner, that the tendency is not to the disease itself, but to conditions which predispose to it.

Although, however, we may not admit heredity in its most absolute sense to be a cause of emphysema, it does not follow that what, in the absence of precise knowledge, we call the 'constitution' of the patient has no influence in determining its occurrence. The tone of muscle and its capacity for energy vary enormously in different individuals, though no structural differences can be demonstrated; and the same may be true of the elastic tissues. That such is the case is certainly possible, and, in the opinion of the writer, probable; and, if so, the occurrence of dilatation of the pulmonary alveoli may well be brought about in certain individuals by a degree of increased tension within the air passages, such as accompanies ordinary straining efforts, which we are not accustomed to regard as adequate for the production of emphysema, and which are not adequate in persons of firmer fibre.

All who have studied the subject of emphysema from a clinical standpoint must have met with cases in which the ordinary exciting causes of the disease have apparently been absent. In many of these the absence has, it is true, been apparent only, for it is difficult to realise how slightly a chronic winter cough impresses itself upon the memory of some patients; hospital patients, indeed, rarely mention such an ailment unless directly questioned about it. But due weight having been given to this source of error, there undoubtedly remains a certain small proportion of cases in which no adequate exciting cause can be discovered. This lack of resisting power on the part of the elastic tissues of the lung may certainly be acquired, it may possibly be inherited, and is probably a common result of the degenerative processes incidental to advanced age. A case recorded by Hugner proves clearly that, after recovery from an attack of pneumonia, emphysema of the affected part may ensue upon the resumption of an occupation, that of a cornet player, which favours the occurrence of the disease, but which had been previously followed without injury to the lungs.

Structure of the pulmonary lobules.—A lobule of the lung may be regarded as a lung in miniature; a clear idea of the structure of a single lobule will therefore enable us without much effort to construct the whole organ.

Each lobule, more or less cone-shaped, is surrounded by areolar tissue; at its apex the lobular bronchus, the blood vessels, lymphatics, and nerves unite to constitute it. The bronchus, after a short course within the lobule, divides and subdivides, with at first but slight diminution in size, forming passages which are termed the interalveolar or intervesicular passages. The course of the bronchus is at first fairly straight, but as the divisions increase in number and diminish in size the direction constantly changes. As the alveolar passages approach the surface of the lobule they cease to diminish in size. Each passage beyond the final division terminates in a blind extremity, which, if not dilated, often appears to be so, from the fact above stated, that the passages do not diminish in diameter. In some cases, however, the ends of the alveolar passages are really dilated, and from this appearance the name 'infundibula' has been applied to them, but a distinctive name is scarcely necessary. As the bronchus enters the lobule rounded orifices appear upon its walls. These are the openings of the alveoli, which may be regarded as the radicles of the bronchial tree. They are at first but few in number, but gradually increase. As the air channel passes onwards through the lobule, and the interalveolar passages are formed, their walls become more and more thickly studded with the orifices of the air vesicles, until, by the time the surface of the lobule is reached, the blind ends of the passages are found to consist entirely of the orifices of these small recesses.

From the foregoing description it will be seen that the air vesicles of the terminal passages open into a common space, adjacent vesicles being separated by incomplete partitions, and that all the air cells of a single lobule are, to a considerable extent, confluent one with another. Adjacent interalveolar passages are separated by partitions formed at the site of branching of the air vessels.

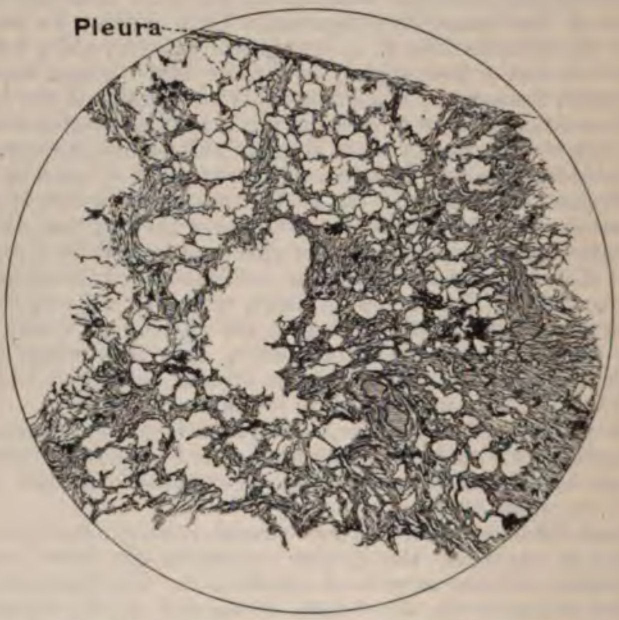


FIG. 60.—EMPHYSEMA OF THE LUNG (× 7 diameters)

Showing spaces of various sizes produced by dilatation of alveoli. The larger spaces are formed by coalescence of smaller ones. Pigmentation and some degree of fibrosis is also present.

The interalveolar passages and their terminations are chiefly composed of unstriped muscular fibres, arranged circularly, and supported by a delicate fibroid tissue mingled with elastic fibres. The walls of the air vessels consist of a delicate membrane crossed by a network of elastic fibres.

The capillaries on the terminal passages are covered by epithelium only on the surface looking towards the cavity; those in the septa project into the cavities on either side.

Morbid anatomy.—The primary lesion in emphysema consists in an enlargement of the terminal interalveolar passages, which increase in size at the expense of the alveoli opening into them. Sometimes, however, the alveoli appear to be the first to undergo dilatation. In any case the effect is, by pressure and stretching, to diminish the blood-supply to the epithelial and vascular structures in their walls. The alveolar epithelium undergoes fatty degeneration, the granules being aggregated round the remains of the nuclei. The septa between adjacent alveoli are reduced to small projections by a gradual process of wasting; subsequently the partitions between neighbouring alveolar passages are perforated, and they become fused into rounded spaces, the size of which tends to increase with the continued operation of the exciting cause of the disease. It is obvious that this process must be accompanied by a great destruction of the pulmonary capillaries, an important factor in determining some of the effects of the disease. According to Rindfleisch, wide communications are formed between the pulmonary artery and the pulmonary and bronchial veins, thus relieving the tension in the former vessel, but allowing the blood to pass through the lungs without undergoing proper aeration.

Varieties of emphysema.—Certain varieties of the disease may be recognised both clinically and pathologically; the morbid changes by which they are characterised will now be considered.

Large-lunged emphysema (*chronic hypertrophic emphysema*). The objection to the term 'hypertrophic' as applied to this condition is that its use connotes increased functional activity, whereas in emphysema the opposite condition prevails. The term here adopted, which was first suggested by Jenner, appears preferable, as it expresses an absolute fact and involves no theory.

When the thorax is opened the lungs not only fail to collapse, but remain fully distended, and, when the smaller bronchi have been obstructed from inflammation, may even bulge forward. The apices fill the supraclavicular regions, and the enlarged anterior margins may be in contact beneath the whole length of the sternum, the precordial area being occupied by the distended auricular process of the left upper lobe. The diaphragm is depressed owing to the permanently inflated condition of the lungs. After removal, when the organs are held with the base upwards, the distended and rounded edges of the lower lobes form the sides of a deep cup.

The lungs in emphysema were likened by Laennec to a pillow of down, and the simile can scarcely be improved upon. They are soft and non-crepitant; when compressed a deep pit forms and remains. They are pale grey in colour, and are marked by black pigment, scattered over the surface in lines and spots, the lines in some cases mapping out the lobules. On close inspection the superficial portions have the appearance of a very fine froth, consisting of the most minute air bubbles covered by the pleura. This is rendered more obvious by the use of a hand lens.

In some cases large rounded bullæ containing air are present, usually along the anterior margin of the upper lobes or around the bases, but they may be absent when the disease is advanced and widely disseminated. Some are attached to the lung by a narrow peduncle only, the auricular process of the left upper lobe being a common site of this particular lesion. They collapse when opened, and delicate fibrous bands, the remains of alveolar septa and obliterated vessels, may then be found crossing the interior.

These two forms, the 'local' or 'bullous' and the 'general,' are too frequently associated to justify a separate nomenclature, but it is important to bear them in mind, as will appear when we come to consider the physical signs of the disease.

On section the lungs are bloodless and dry, except perhaps at the bases, where œdema may be present. This, however, pertains more to some complication, such as bronchitis or cardiac failure, and is no necessary effect of the disease.

If the section be made from the extreme posterior margin forwards, the portion of the lung which occupies the hollow beside the spine will often be found in an advanced condition of emphysema, large spaces being present beneath the pleura, and extending for perhaps half an inch or more into the lung.

The smaller bronchi are in some cases dilated to a slight degree, but bronchiectasis is by no means frequently associated with emphysema.

Atheroma of the pulmonary artery is commonly present, and in advanced cases patches may be found throughout the vessel, not even the smaller branches escaping. It is a result of the increased strain on the walls of the vessel from the obstruction to the passage of the blood through the lungs. There is very often a complete absence of pleural adhesions, a condition rarely observed in adults unless they are the subjects of emphysema.

Small-lunged emphysema (syn., *senile atrophic emphysema, senile atrophy of the lungs*).—The most striking clinical and pathological characteristics of this condition of the lungs are indicated by its name. It appears to be primarily an atrophic change, incidental to advanced age, and shared by the lungs equally with the other organs of the body. Its title to be considered either as a substantive disease of the lungs or as a distinct variety of emphysema is doubtful. It never occurs apart from a general condition of atrophy, and the slight degree of emphysema which accompanies it is probably induced by the cough of a bronchial catarrh, from which the very aged are rarely quite free. It is, however, convenient and in accordance with custom, to describe it as a variety of emphysema. The subjects of senile emphysema present a wasted, shrivelled, and withered-up appearance: the thorax is rigid, the space within is small, the lower ribs are almost in contact and very obliquely placed. On opening the chest the uncovered area of the heart is not diminished, it may even be enlarged; the lungs readily collapse, falling back towards the spine; they are smaller than normal, deeply pigmented, almost black in colour; light, dry, and easily compressible. On section they present a coarsely reticulated structure. The vesicles are enlarged by a process of fusion, the result of wasting of the septa, and this change may in places be so advanced as to involve adjacent lobules. Large bullæ are rare, but the margins are in some cases much dilated. The bronchi are thin-walled, and have undergone dilatation, the lining membrane is commonly inflamed, and the tubes contain puriform fluid. Collapse and œdema are often present, and are generally most marked on the posterior aspect of the lower lobes.

Local emphysema—compensatory emphysema.—This form of the disease is invariably secondary to some pulmonary lesion, most commonly to tuberculosis which has undergone either complete or partial arrest. In the presence of a contracting lesion within the lungs—for instance, a cavity or an area of fibroid tuberculosis—either the surrounding tissue becomes emphysematous, or the pleura thickened, the result being determined by the nature, site, and extent of the lesion. In the case of a lesion situated close to the surface, if the lung intervening between it and the pleura is condensed, airless, and incapable of expansion, the visceral and parietal layers of the pleura, partially united by fine fibrous bands, tend to become separated. The space is at first filled with yellow serous exudation, which ultimately undergoes transformation into a thickened fibroid tissue almost cartilaginous in density. The apex of the lung, in cases of very chronic pulmonary tuberculosis, when the upper lobe is almost completely occupied by a contracted thick-walled cavity, shows such a thickening of the pleura as is here described. If, on the other hand, the lung tissue around the lesion is not the seat of such advanced changes, and still admits of the entrance of air, the surface vesicles enlarge, coalesce, and form bullæ sometimes of considerable size. Such a condition is commonly seen at the apex of the lung, and is a certain guide to a contracted lesion within. The surface may be scarred and puckered, and on section dense pigmented fibrous bands are seen surrounding old fibrous, caseous, or calcareous lesions, and extending into the neighbouring emphysematous tissue. The vessels and bronchi in such an area are usually obliterated, but on its confines the latter may be found dilated.

Another common site of local emphysema is the posterior and upper part of the lower lobe. Here the change is secondary to a contracting lesion, usually a cavity, at the apex of the lung, and may occupy a considerable area. In one such case observed by the writer the posterior aspect of the contracted upper lobe was completely covered by the upper part of the lower lobe. No bullæ are formed, but on section a coarsely reticulated structure is seen, replacing the normal tissue and reaching downwards along the posterior aspect of the lobe.

In cases of fibroid transformation of tubercle the densely pigmented contracting fibrous nodules are often found embedded in emphysematous lung, the whole presenting appearances which show unmistakably that the fibrosis has preceded the emphysema.

Acute vesicular emphysema.—The definition of the disease given at the head of this article does not include a lesion consisting merely in an over-distension of healthy alveoli, such as is present in the above-named condition. Moreover atrophy of the alveolar walls is an essential part of the morbid anatomy of emphysema, but is not met with in acute vesicular emphysema, which on these grounds also cannot be considered as a true variety of the disease. Acute vesicular dilatation is sometimes found after death from acute bronchitis, or from asphyxia, when this has been accompanied by violent inspiratory efforts; or when, from collapse or other cause, the air has been prevented from entering portions of the lung, thus throwing an increased strain upon the alveoli of other parts.

It may be demonstrated, however, by physical examination that a similar condition is present in cases which are not fatal, and also that after a time the lungs return to their normal size, a proof of the absence of structural change

The lung in such a conditi nsion is large and pale, and with a hand lens the incr the surface alveoli can be readily seen.

Lesions associated physema.—*Lungs.*—Although, in the majority of cas chitis and emphysema stand related to one another as caus effect, it is nevertheless true that when emphysema has become established it increases the tendency to bronchitis.

The over-distended air vesicles compress and obstruct the capillaries and impede the circulation through the pulmonary and bronchial vessels. The bronchial mucous membrane becomes congested, and the condition thus established greatly increases the liability to inflammatory attacks. Rupture of dilated vesicles may lead to pneumothorax, but if the pleura overlying the site of rupture remains intact, interlobular emphysema results. Death is rarely due to pneumothorax so caused, but one such case has been observed by the writer, and others are on record.

Bronchi.—As already described, the bronchi are often found obliterated and forming thin fibrous bands in large emphysematous bullæ; they are, however, occasionally, but not commonly, found dilated to a moderate degree in less advanced cases of general emphysema, and more often in localised emphysema. In the atrophic form the bronchial walls are usually thin; in other forms they may be somewhat thickened, as may also be the walls of the vesicles and interalveolar passages.

Heart.—The obstruction to the flow of blood through the capillaries of the lungs naturally increases the tension within the pulmonary artery and requires a more forcible contraction of the right ventricle. This leads to hypertrophy of the ventricle, and thus for a time equilibrium may be restored. But when, from any cause, the structural integrity of the new muscular tissue is impaired, particularly if at the same time greater stress is thrown upon the right ventricle, dilatation follows, the tricuspid orifice enlarges, and the valve becomes incompetent.

The right auricle, probably already somewhat enlarged, now undergoes still further dilatation, and the superior and inferior venæ cavæ are similarly affected. Congestion of all the organs which are drained by the systemic veins necessarily follows. The portal system may become involved at a later period. This sequence of events is not uncommonly initiated by an attack of bronchitis.

The dilatation and hypertrophy of the right ventricle, including the conus arteriosus—for the latter is always involved—are usually found on autopsy to be associated with similar but less advanced changes in the left ventricle, a result probably due, at least in part, to their intimate association both in structure and functional activity.

Fatty changes are not uncommonly observed in the heart in emphysematous subjects, and are due to the impaired nutrition of the muscular walls from obstruction to the return of blood by the coronary veins.

As a result of the enlargement of the lung and the permanently depressed state of the diaphragm, the position of the heart becomes altered. It lies lower in the chest, and its axis is more nearly horizontal. The front of the heart is formed entirely by the enlarged right ventricle and auricle. The altered position and size of the organ account for the pulsation commonly observed in the epigastrium in well-marked cases of emphysema; but of these two factors the change of position is the more important.

Secondary changes of a fibroid character are not infrequently found in the tricuspid and mitral valves, and more rarely in the aortic valve also.

Liver.—The changes in the liver resulting from chronic venous congestion are too well known to require complete description. The organ is enlarged and the hepatic veins are dilated. The section presents the 'nutmeg' character, and there is some degree of induration; but emphysema alone is as powerless as chronic mitral disease to produce a true cirrhosis.

The *kidneys* may be enlarged and cyanotic, but in a considerable proportion of cases they are granular from the presence of chronic interstitial nephritis, a disease with which emphysema is not uncommonly associated. The *spleen* is as a rule enlarged and hard, but its condition varies.

Chronic venous congestion of the *stomach* may give rise to catarrh and hæmorrhage into the mucous membrane. The *brain* also shows evidence of venous congestion.

As considerable differences exist in the symptoms and physical signs which characterise the various forms of emphysema, it is necessary to describe them under their respective headings.

Symptoms of large-lunged emphysema.—The symptoms strictly referable to emphysema are very few, the condition, apart from its complications, being one of which patients have little or no knowledge, and one of which therefore they rarely complain.

Dyspnœa is the most important symptom, but even this is seldom mentioned until it has become somewhat urgent. It is in proportion to the extent of the disease. At first slight, and only experienced on exertion, especially on walking uphill, it may spiration owing to the non-expansion of the emphysematous lung. Bulging of the spaces may be well marked when the patient coughs. The respiratory movements are restricted, and the expiratory act is much prolonged notwithstanding the forcible contraction of the abdominal muscles. The gradual expansion of the chest during inspiration, which is characteristic of health, tends to be replaced by a uniform upward lift, during which the accessory muscles of inspiration stand out in strong relief. In some cases, however, the infra-axillary regions are drawn inwards and the sternum projected forwards, whilst at the same time the epigastric region, instead of bulging during inspiration, may be visibly depressed. This recession of the lower ribs during inspiration is often well marked and may accompany the deformity of the chest called the 'transversely constricted' thorax, which is usually a relic of infantile rickets.

The downward and axial displacement of the heart, combined with the hypertrophy and dilatation of the right ventricle, to which reference has already been made, are jointly the causes of the epigastric impulse commonly observed in emphysema.

A horizontal sulcus is sometimes observed to extend across from side to side about the level of the lower part of the costal arch.

A broad line of dilated venules is often seen in emphysematous subjects tending obliquely upwards on either side along the line of the lower costo-chondral junctions, and across the base of the ensiform cartilage, and therefore corresponding roughly with the attachment of the diaphragm. It is rarely complete posteriorly. It is not limited to cases of emphysema.

Palpation.—The vocal fremitus is diminished. The impulse in the precordial area is generally feeble owing to the cushion of lung intervening between the heart and the chest wall; but the hypertrophied right ventricle, in the absence of much enlargement of the lung, may cause a heaving impulse in the lower sternal region.

Percussion.—A hyper-resonant note will be found in regions, such as the precordial and hepatic, which are normally dull; or dulness may still be present, but over a much diminished area, whilst behind it is by no means uncommon to find well-marked resonance as low as the twelfth rib. Inspiration and expiration make but little change either in the area of resonance or in the pitch of the note on percussion.

Auscultation.—The character of the respiratory murmur varies with the form of the predominant lesion, whether this be of the bullous type or general in its distribution. If 'bullous,' the breath sound is weak over the sternum and along the margins of the upper lobes, but harsh beneath the outer half of the clavicle, whilst in the 'general' form the breath sound over the upper lobes is everywhere feeble. It is right to state, however, that the opposite opinion is held by some authorities. In place of the normal vesicular murmur audible on inspiration, the continuous low-pitched rumbling sound produced by the contraction of the muscles is often very distinct.

When the disease is fully established the expiratory sound is almost invariably prolonged, often very markedly so; in fact, during an intercurrent bronchial catarrh, its duration may be so prolonged as to be nearly four times that of inspiration.

These changes in the respiratory sounds are usually most obvious over the upper part of the chest, but when the posterior aspect of the lower lobes is affected the breathing will be weak at the bases, and fine crackling râles may be present there also. These signs are important both as evidence of advanced disease and of œdema of the affected parts of the lung.

At the apex of the heart the sounds are feeble, the characters of the first sound being determined by the relative preponderance of hypertrophy or dilatation of the right ventricle. In the former case it is low-pitched and prolonged, in the latter short and sharp, but weak. The point of maximum intensity of the sounds at the base is lower than normal, and, owing to the increased tension in the pulmonary artery, the second sound is accentuated, and may be reduplicated.

A rough murmur is often audible in cases of emphysema about the sternal end of the sixth left interspace and over the seventh rib, close to the base of the ensiform cartilage. It is systolic in time, usually short, sharp, localised, and superficial, and it often resembles more nearly a rough reduplication of the first sound than a murmur. It may be due to a 'white patch' on the anterior surface of the right ventricle, a condition often present in emphysema. The effect of change of position of the body on this sound is variable. It may disappear or remain unchanged. The only importance of the sign arises from the fact that it is very likely to be mistaken for the murmur of mitral regurgitation.

Symptoms of small-lunged emphysema.—In this form of the disease the symptoms are much less pronounced. The most important change in the lungs, the atrophy, is but a part of a general process of wasting in which all the tissues of the body, including the blood, share alike. The respiratory needs are therefore less, and they may be adequately met by a smaller pulmonary area. The capacity for exertion is limited owing to the feebleness of muscular power; and, in the absence of effort, there may be little or no dyspnœa.

Another point of difference from the variety just considered is that atrophic emphysema is rarely complicated by attacks of bronchial asthma; but intercurrent bronchitis may induce dyspnœa which, although differing in its mode of onset, is hardly less in degree than that which characterises the asthmatic paroxysm.

Physical examination; *inspection.*—The emaciated and withered appearance of the subjects of the disease has already been mentioned. The evidences of venous obstruction, such as cyanosis and clubbing of the fingers, are absent; as also are the effects which that condition produces in the size, shape, and position of the heart. The chest assumes the barrel shape as a result not of a process of enlargement, but of 'shrinkage' in all its diameters, and especially in the lateral. The gradual diminution in the size of the lungs is necessarily accompanied by a recession of the ribs, which assume a more oblique position. The interspaces from the first to the fourth on the front of the chest are often both wide and deep; but the increased obliquity of the lower ribs tends to approximate them, so that the interspaces may be obliterated, or adjacent ribs may even overlap each other.

Inspiration is shallow, the rigid thorax moves as a whole, the upper interspaces recede, and descent of the diaphragm is limited.

Percussion.—The note is hyper-resonant, but it tends to be more clear in tone and tympanitic in quality than in large-lunged emphysema. The area of precordial dulness is not diminished and may possibly be increased. The hepatic dulness is also diminished.

Auscultation.—The breath sound is weak, but the expiratory sound is not prolonged to nearly the same extent as in the large-lunged variety. Adventitious sounds are not necessarily present, but the coexistence of chronic bronchitis is so common as to make their complete absence very rare; fine and medium bubbling râles may be heard over the bases of both lungs. Fine crackling râles may be audible over the same area if œdema is present.

Other pulmonary complications will give rise to the auscultatory signs by which they are usually characterised, modified to some extent by the presence of emphysema.

Symptoms of localised emphysema.—On reference to what has been stated as to the mode of production and common sites of this variety of emphysema, it will be seen that the symptoms must necessarily depend upon the condition to which it is secondary. It may, however, be repeated that it is frequently a sequence of tuberculosis, and its presence at the apex of a lung should suggest the possibility of such a connection.

An enlargement of one lung or of a portion of it, secondarily to disease and contraction of the opposite lung, is not necessarily due to emphysema; it may be a true hypertrophy. The test by which the two conditions are distinguished is that of functional activity. If this is increased the enlargement must be regarded as hypertrophy; if diminished, as probably due to emphysema: in the former case the breathing is puerile, in the latter it is usually feeble with prolonged expiration.

Symptoms of acute vesicular emphysema.—As already stated, this condition is only recognised as a form of the disease in deference to tradition.

It originates during a state of extreme dyspnœa, the urgency of which it doubtless increases; but the result to the patient is probably determined almost invariably by the nature of the exciting cause and not by the effect produced upon the lungs. Cyanosis is very likely to be present during the attack.

The chest will be in a condition of extreme inspiratory distension. The nature of the breath sounds and adventitious sounds will vary with the exciting cause.

The **diagnosis** of the large-lunged form of emphysema rarely presents much difficulty. It is suggested by a history in which cough and dyspnœa are prominent features, or by the patient being engaged in some occupation known to involve severe muscular effort; it is confirmed on examination by the alteration in the form of the chest, the hyper-resonance on percussion, diminished movement, and feeble respiratory sound—signs which are present on both sides of the chest.

Error has apparently arisen at times from pneumothorax being mistaken for this form of emphysema. In such cases the methodical examination of the chest has probably been neglected, and reliance placed upon one step alone in the process, possibly on percussion. In pneumothorax the enlargement of the affected side, the obliteration of the interspaces, the absence of movement, contrasting with the increased movement of the healthy side—if it be healthy—the displacement of the heart to the sound side, the more amphoric note on percussion, and the absence or the amphoric quality of the breath sounds, are signs which combine to form a picture that, in well-marked cases, should be unmistakable.

It is possible, however, for a collection of air, confined to a very small part only of the pleural cavity by firm adhesions, to give rise to signs which may be mistaken for those of emphysema. Such a case, due to the rupture of an emphysematous bulla near the base of the lung, came under the notice of the writer. It is sufficient to mention it as a possibility to be borne in mind without discussing in detail the diagnosis of a condition of such rare occurrence.

Aneurysm of the transverse part of the arch of the aorta compressing the trachea may be mistaken for emphysema with bronchitis. The tracheal stridor and brassy cough, the dulness, or at any rate the absence of increased resonance over the manubrium, and the loud tracheal breathing over the same area, usually suffice to prevent error.

'Emphysema and bronchitis' is occasionally the diagnosis on admission to hospital of cases in which the primary disease is really stenosis of the mitral orifice; cardiac failure, pulmonary engorgement, and œdema have supervened, and the murmur has disappeared. After a few days of rest and treatment considerable improvement as a rule takes place, the murmur again becomes audible, and the true nature of the case is then obvious.

True cardiac dyspnœa is distinguished from that accompanying emphysema by its 'panting' character; but failure of the right heart often follows upon long-standing emphysema, and the dyspnœa is then the resultant of the two conditions and partakes of the characters of both.

An examination of the sputum for tubercle bacilli should always be made in cases of emphysema and bronchitis, particularly in such as are accompanied by marked emaciation. In the fibroid form of pulmonary tuberculosis, which is often associated with emphysema (not so-called 'fibroid phthisis'), bacilli may be absent and the true nature of the disease may only be discovered on autopsy. The absence of pyrexia in such cases is not a distinguishing symptom of much value, fibroid tuberculosis being often unaccompanied by fever, at any rate for intervals of considerable duration.

The diagnosis of the atrophic form of emphysema is but rarely attended with difficulty.

Prognosis.—True emphysema—that is, dilatation with atrophy—is a permanent condition, with a decided tendency to advance. But whether it increase, and if so, at what rate, depends chiefly upon the continuance of the exciting cause, which, in the great majority of cases, is the cough of catarrh or bronchitis. If the patient is able, by change of residence, or in other ways, to shield himself from adverse conditions of climate, the disease may remain stationary. Under any circumstances its course is chronic, and life only becomes endangered when complications arise.

The extent of the lesions will naturally influence the prognosis; but the effect produced upon the heart and circulation is a far more important factor in determining the probable duration of life. As dyspnœa is the chief evidence of this effect, its degree during rest and on exertion becomes one of the main elements in prognosis. The condition of the veins of the neck as to over-distension, pulsation, and filling from below, are important guides to the state of the right side of the heart.

The existence of enlargement of the liver, œdema of the legs, ascites, and albuminuria marks an advanced stage of cardiac failure.

The presence of renal complications, particularly chronic interstitial nephritis, is of especial importance in prognosis.

Treatment.—Sufferers from emphysema rarely ask for advice on that ground alone, the disease being one of the existence of which the laity may be said to be ignorant. As a rule no complaint is made of the accompanying dyspnœa, to which the patient has become so habituated that he has ceased to regard it. In the majority of cases the condition is discovered when an intercurrent attack of bronchitis leads to an examination of the chest.

Atrophy of the alveolar walls, destruction of the capillaries, and wasting of the elastic tissues, are changes which cannot be repaired, and a return to the normal state is only possible in the cases of temporary over-distension which occur for the most part in young subjects, as a result either of laryngeal obstruction, spasm, or whooping cough, or of bronchitis accompanying an acute disease, such as measles.

Much, however, may be done to stay the progress of the disease by shielding the patient from further attacks of bronchitis, or by advising a cessation of any occupation which necessarily involves a strain upon the respiratory organs. Treatment may also usefully be directed towards the relief of the secondary effects upon the heart and circulation.

Emphysema once established undoubtedly predisposes to bronchitis; it is therefore of the first importance that all known causes of catarrhal inflammation should be carefully avoided. Those whose means permit will be well advised to spend the winter and spring in a warmer climate than is to be found in this country at such times; many sufferers, however, although they know this full well, are prone to delay their departure unduly, and an early November fog finds them still here; the result too often is a severe attack of bronchitis and much increase in the emphysema. Persons who are unable to leave home, if they hope to escape an attack of bronchitis, must exercise the greatest care in avoiding cold north and east winds, foggy and damp air, over-fatigue, or sitting in draughty rooms, and anything likely to give rise to a chill. Notwithstanding the unsightly appearance, a respirator, or woollen 'comforter' covering the mouth, by warming the incoming air, is of real service in warding off attacks of bronchial catarrh.

The conditions which give rise to increased tension within the air passages have already been described; it will be sufficient, therefore, to state that it is absolutely necessary for the sufferer from emphysema to avoid them if he wishes to escape an increase of his disease.

The effect upon the respiration is a useful test as to whether any form of exercise is harmful either in kind or degree; if it causes dyspnœa it should be avoided. The bowels should not be allowed to become confined, as, in addition to the gastro-intestinal derangements likely to ensue, much harm may be done by straining efforts in defæcation.

In the work of Dr. Williams on Aero-therapeutics a full description will be found of the various forms of apparatus used in the application of condensed air to the body as a whole, and of condensed or rarefied air to the respiratory surface. Notwithstanding that much has been done in recent years to render our knowledge of this branch of treatment more exact, it is still but little used in this country. This is doubtless due to the fact that patients are rarely under treatment for emphysema apart from its complications; and also to the small number of compressed air baths available for use.

The condition of the lungs in emphysema indicates that expiration into rarefied air should afford relief. This proceeding causes a diminution in the amount of residual air, and an increase in the volume of inspired air: thus a partial retraction of the lungs and a rise in the position of the diaphragm are brought about. These changes are accompanied by a lessened circumference of the chest, and by an increase of the vital capacity and of the force of inspiration and expiration. The apparatus of Waldenburg is most suited for this form of treatment. Expiration into rarefied air produces a sense of extreme constriction within the chest and certainly diminishes the amount of residual air. The 'vital capacity' of patients with emphysema under treatment by this method undoubtedly increases; but this fact cannot be accepted as an absolute proof of its value, as it also follows the use of the apparatus by those whose lungs are structurally sound, practice enabling the individual to produce a better result. The benefits to be obtained from expiration into rarefied air are, however, much less marked than those which attend the use of compressed air applied to the body as a whole.

The writer has given a prolonged trial at the Brompton Hospital to the use of the compressed air bath in the treatment of emphysema associated with bronchitis, and is able to endorse the favourable opinions expressed by Dr. C. Theodore Williams and others as to its great value.

Patients almost invariably state that they breathe more freely whilst in the bath, and after a considerable number of baths (from twenty to thirty or more) have been taken this feeling becomes continuous and has remained whilst the patients have been under treatment. The greater capacity for exertion which follows the use of compressed air baths in emphysema has been tested by observation of the gradually increasing facility with which patients thus treated have been able to mount a flight of one hundred and twenty steps which leads from the basement where the bath is situated to the 'gallery' (wards) occupied by them. Patients who at first were obliged to use the lift to return to their ward or were only able to climb the stairs with many halts to take breath, have been able gradually to reduce the number of stoppages on the ascent, and many have finally been able to return from the basement to the topmost floor without stopping once.

In addition to the greater freedom of respiration and increased capacity for exertion, the cough becomes less frequent and the quantity of expectoration is reduced.

It is not quite clear how these favourable results are produced. In a healthy person the effect on the respiratory organs of submitting the body as a whole to air gradually condensed to the extent of three-sevenths or one-half an atmosphere is to cause diminished frequency of respiration, enlargement of the lungs, increase of the vital capacity, and probably also an increase in the amount of oxygen absorbed. The change is attributed to the greater density of the air, and consequently to the increased amount of oxygen supplied to the lungs. The respiratory power and the elasticity of the lungs, both during and after the bath, are increased, the chest is enlarged in all its measurements, and the diaphragm assumes a lower level. In the subjects of emphysema, however, the effect of the bath is to cause a reduction in the size of the chest, as ascertained by measurement of the circumference, and also in the amount of distension of the lungs, as proved by the reappearance of dulness in the precordial and hepatic regions. The diaphragm is raised instead of being lowered, and epigastric pulsation may be replaced by an impulse more nearly in the normal situation of the apex beat of the heart.

It appears probable that the condensed air penetrates parts of the lungs which have been long unused in respiration, and in which air has been, so to speak, imprisoned at a high tension, the escape of this air is facilitated and contraction of the lung follows.

In some cases the improvement following the use of the bath is only temporary, and in cases of emphysema accompanied by asthma the writer has observed very severe attacks of dyspnœa to follow very shortly after a bath. If this should occur after the second bath it is generally better to discontinue its use. Many cases of asthma are, however, greatly benefited by this method of treatment.

The treatment of an attack of bronchitis occurring in a patient the subject of emphysema is not materially modified by the complication; but the duration of the attack is sensibly prolonged, and the danger to life is much greater, owing to the loss of power of expectoration which results from the diminished elasticity of the lungs.

Spasmodic dyspnœa often accompanies such an attack, and requires the use of such remedies as stramonium, lobelia, belladonna, grindelia, or iodide of potassium in large doses, in addition to the ordinary drugs used in the treatment of bronchitis. The desirability of employing morphia in such cases will depend chiefly on the relative preponderance of the spasmodic or the catarrhal factor. The nearer the attack approaches in character to one of true asthma the greater is the probability of relief from a subcutaneous injection of morphia; whilst on the other hand if the dyspnœa is chiefly due to the accompanying bronchitis, the use of morphia may be attended with the greatest danger. The history of previous attacks, the mode of onset, the presence of pyrexia, the character of the adventitious sounds—for instance, the presence of fine or medium bubbling râles, indicative of an affection of the smallest bronchi or of the alveoli—and particularly the condition of the bases of the lungs, are some of the points to be considered in determining such a question. In the treatment of the attacks of wheezing, so often met with in emphysema apart from any serious bronchial attack, a stimulating liniment containing turpentine and iodine rubbed into the chest is often of much service. Iodide of potassium in doses of five, eight, or ten grains three times daily, in combination with extract of stramonium and carbonate of ammonia, generally affords relief. In the intervals of comparative freedom from such attacks, and often throughout the winter months, the administration of cod liver oil is of hardly less service than in cases of pulmonary tuberculosis. It is of special benefit when nutrition is failing, as is commonly the case in advanced stages of the disease, and in the atrophic emphysema of the aged. Iron in combination with spirits of chloroform is often taken by patients with emphysema with much benefit.

Turpentine, terebene, and balsamic remedies are of service where expectoration is excessive; this symptom is, however, due to the accompanying bronchitis, and its treatment is described in the chapter on that subject.

Cyanosis is an indication for venesection, and the necessity is
t when there is evidence of great over-distension of the right
f the heart, with tricuspid regurgitation, pulsation in the
r veins, and œdema of the feet. Digitalis should be given as
the blood has been removed; and its use may be necessary
which are not so advanced as to require venesection.

n, as is not uncommonly the case, emphysema supervenes
chitis of gouty origin, the existence of that factor in the case

Female, æt. 5. Diphtheria; tracheotomy not performed. Emphysema of root of neck; mediastinal, interlobar, and interlobular emphysema; pneumothorax (R); pulmonary collapse.

Male, æt. 5. Diphtheria; tracheotomy. General emphysema of subcutaneous cellular tissue of neck, trunk and arms; lungs almost completely collapsed from double pneumothorax, air in mediastinum and around roots of lungs; membrane on tonsils and in larynx, trachea, and large bronchi.

Male, age 2 years. Diphtheria; tracheotomy. Larynx completely blocked with membrane, which extended throughout the trachea and main bronchi; lungs collapsed in patches; emphysema of anterior mediastinum.

Male, age 11 years. Diphtheria; tracheotomy. General emphysema; membrane in trachea and bronchi of left lung, latter collapsed; marked emphysema of anterior mediastinum.

Female, age 4 years. Diphtheria; tracheotomy. Interlobar emphysema on right side; air in anterior mediastinum; membrane as far as secondary divisions of bronchi; numerous areas of pulmonary collapse.

The preceding cases illustrate the lesions commonly found in association with interlobular emphysema when that condition occurs in diphtheria, the most important being general emphysema, pneumothorax, and pulmonary collapse.

Symptoms.—In all the cases above described in which tracheotomy was performed there would necessarily be urgent dyspnœa at the time the trachea was opened. The dyspnœa would then be relieved, but the occurrence of mediastinal and interstitial emphysema is accompanied by an increase in the dyspnœa. Should pneumothorax supervene, the difficulty of breathing becomes extreme.

Double pneumothorax is always quickly followed by death.

The breath sounds would almost certainly be weak or absent if the connective tissue of the lung were infiltrated with air. Pneumothorax would be characterised by its ordinary physical signs.

Interlobular emphysema is rarely recognised during life. It may be suspected when subcutaneous emphysema is present, or when pneumothorax occurs. The latter is a serious complication. It is probable that the condition here described is often present but is unsuspected, and that the air is absorbed when recovery takes place.

No definite **treatment** can be adopted for the condition.

J. K. F.

CHAPTER XIV

ASTHMA

	PAGE
Varieties	183
The asthmatic paroxysm	184
Exciting causes of the paroxysm	186
Central	186
Peripheral	186
Pathology	187
Etiology of the disease	189
Diagnosis	190
Prognosis	190
Treatment	191

Definition.—Asthma is, as a disease, characterised by paroxysmal attacks of dyspnœa of a peculiar type.

The term, qualified by a descriptive adjective, is still in ordinary medical language applied to a variety of conditions which, although differing in origin, present a superficial likeness to one another, owing to the fact that all are accompanied by attacks of difficulty of breathing. Although this use of the term is to be regretted, it cannot well be ignored, and a brief definition of each variety may possibly help to a clear understanding of the subject. These are:

(A) True *spasmodic asthma*, in which a nervous factor is the most marked feature of the disease, other changes being either absent or secondary.

(B) *Bronchial asthma*, a variety of the disease in which paroxysmal attacks of dyspnœa of the true asthmatic type occur in an individual previously the subject of bronchitis and emphysema.

(C) *Cardiac asthma*, an affection characterised by attacks of dyspnœa occurring in the subjects of valvular or myocardial disease.

(D) *Renal asthma*, a condition sometimes present in uræmia from acute or more frequently chronic nephritis.

(E) *Hay asthma*, a variety of true asthma in which, in the majority of cases, the stimulus inducing the attack is the presence on the mucous membrane of the air passages of the pollen of flowering plants, particularly of grasses.

is small and feeble, and may intermit with inspiration (puls paradoxus).

When the patient is almost exhausted, the paroxysm begi slowly to pass off. Cough occurs, secretion is increased, a pellets of semi-transparent mucus, pale grey in colour, and liken to boiled tapioca, are expectorated. The patient may then fall aslee In the expectoration following the decline of a paroxysm, Cursc mann's spirals and Charcot-Leyden crystals may generally be fou in the tough pellets above described. The former (see fig. 61) a

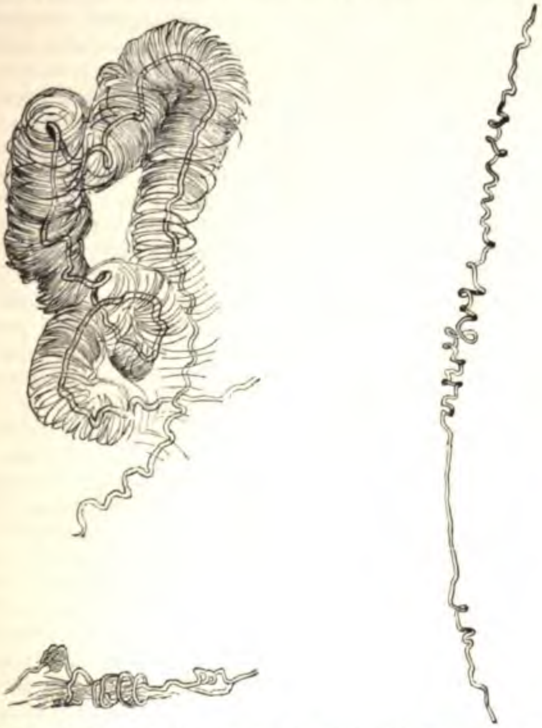


Fig. 61.—Curschmann's Spirals of different Form and Size ×90

spiral threads of mucin having the form of a corkscrew, or central thread contained within a spiral coil. They are not peculi to asthma, having been found in the sputa of cases of bronchi and pneumonia. The spirals are probably formed in the minu bronchi. Charcot-Leyden crystals are pointed octahedra, resultin it is believed, from the combination of phosphoric acid with organic base. They also have been found in other affections.

Such attacks are of variable duration. They may last from tv ix hours, when the patient may fall asleep, and on waking fi he can breathe freely. More often, however, the breathi continues somewhat laboured for a day or two, and pellets of mucus continue to be expectorated. In severe cases the paroxysm may recur for several nights in succession.

The exciting cause of the paroxysm may be either (A) Central or (B) Peripheral.

A. *Central.*—Emotion may excite an attack in some cases; in others it may terminate it. 'Neurasthenia' is said to be a cause of asthma. Certain 'toxic' causes of the disease, such as the presence of lead in the system, should possibly be placed under this heading, and renal asthma may also be due to a central cause.

B. *Peripheral.*—(*a*) *Nasal.*—The occasional association of *nasal polypus* with asthma has long been known, and also the fact that the removal of the growths may be followed by a cessation of the asthmatic symptoms. Much attention has also of late been directed by Hack of Freiburg and other writers to a *vascular turgescence of the cavernous tissue* covering the turbinate bones during the attacks, and to a chronic thickening of the mucous membrane over the same parts as a cause of asthma. It was at one time even maintained by some that in almost every case these structures required removal, and quite lately the writer met with a lady, aged sixty-three, who had submitted to several operations for this purpose, attended by the outlay of considerable sums of money, but without obtaining relief. Certain *odours* and *scents* and *gases* will produce an attack in some individuals. As examples of this may be mentioned the smell of ipecacuanha, that given off by some animals, and particularly by cats, and the fumes of sulphurous acid and chlorine.

(*b*) *Dental.*—The irritation of teething may in children excite an attack of dyspnœa indistinguishable from true asthma.

(*c*) *Bronchial.*—In the majority of cases the stimulus starts from the bronchial mucous membrane. The influence of *climate* and *locality* is probably felt there; but, as suggested by Dr. Steavenson, it is possible that their influence depends upon 'the electrical condition of the locality taken in relation with the electrical condition of the patient.' Of this, however, there is at present no proof. To some sufferers even one side of a street or the other may mean the difference between comfort and misery. In the case described at the outset, the patient could sleep comfortably in Downing College, Cambridge, but almost invariably had an attack in Addenbrooke's Hospital, close by. Inflammation of the bronchi is one of the most important etiological factors in the disease. An attack may be induced by direct irritation of the bronchi from various causes, such as the presence of dust and fog in the atmosphere. Many cases of true spasmodic asthma, however, are quite unaffected by fog; it appears rather to suit them.

Forcible respiratory movements, such as accompany laughing and sneezing, probably act as excitants by overstimulating the bronchial muscles (Wilson Fox).

(*d*) *Gastro-intestinal.*—Flatulence, constipation, and particularly scybalous distension of the rectum, are somewhat common exciting causes of attacks. Asthma due to the ingestion of some particular article of diet is termed 'peptic.'

(*e*) *Uterine.*—In some cases the reflex stimulus appears to start from the uterus.

(*f*) *Cutaneous.*—Cold applied to the surface, particularly to the feet or insteps, and the disappearance of cutaneous eruptions may be mentioned under this heading.

(*g*) *Pneumogastric.*—Pressure upon the vagus, either from the presence of a neuroma or by enlarged glands in the posterior mediastinum, or an exostosis growing from the vertebræ has been known to produce the disease, but such cases are very rare.

Amongst out-patients seen by the writer at the Brompton Hospital were a considerable number of cases of 'Saturday and Sunday asthma.' Where the state of the nervous system in its relation to the lungs is so sensitive as it appears to be in these individuals, the slightest change from the routine of life, such as occurs at the end of the week, may be sufficient to excite an attack.

Pathology.—We do not purpose to discuss the vexed question of the pathology of asthma in great detail, as that has already been done by many writers, and we would therefore refer the reader for a more complete consideration of this part of the subject to the works of Hyde Salter and Wilson Fox, where it is exhaustively treated.

The asthmatic paroxysm has been attributed to:

(1) Spasm of the bronchi (Floyer, 1720), and later to spasm of the circular muscular fibres of the bronchial wall, after the discovery of their existence, and the proof that their contraction causes narrowing of the tubes (C. J. B. Williams and many other writers).
(2) Spasm of the diaphragm (Wintrich, Bamberger, Lehman, and in part by Traube).
(3) Spasm of the inspiratory muscles (Budd, Kidd, Jaccoud, G. Sée, Steavenson).
(4) Paralysis of the bronchial muscles leading to loss of expiratory power, 'probably in some cases' (Walshe).
(5) Fluxionary hyperæmia of the bronchial mucous membrane, or congestion from extrinsic reflex causes, or vaso-motor neurosis leading to transitory hyperæmia (Weber), analogous to urticaria (Andrew Clark).
(6) Spasm of arterioles and dropsical swelling of the bronchial mucous membrane (Glasgow).
(7) Inflammatory swelling or acute catarrh of the mucous membrane (Bree, Beau, Traube).
(8) Specific microbic inflammation of the bronchial tract (Berkart).

All authorities at the present date admit the presence of a nervous factor in asthma; the unsolved question is whether it acts through the motor branches of the vagus to the muscular fibres of the bronchi, or through the vaso-motor branches of the sympathetic to the vessels of the bronchial mucous membrane. The preponderance of opinion is no doubt in favour of the view that the asthmatic paroxysm is due to a spasmodic contraction of the muscular fibres of the bronchi. We confess, however, that we are unable to arrive at the conclusion that one and one only of these theories must be accepted for all cases of asthma. Having regard to the intimate structural connections of the cerebro-spinal and sympathetic systems generally, and of the vagus and sympathetic nerves in the pulmonary plexuses in particular, it appears to be not only possible but probable that either nerve or both may be affected by a certain morbid condition.

The association of hay-asthma and paroxysmal sneezing—conditions which are accompanied by turgescence of the nasal and bronchial mucous membranes—with asthma is in some individuals and in different members of certain families, so close that it appears impossible to deny that hyperæmia of the bronchial mucous membrane induced through the agency of the vaso-motor centres and nerves may, in some cases, be a factor in the pathology of the asthmatic paroxysm.

The argument that the sudden onset of the attack excludes the consideration of any other factor than muscular spasm appears to have less weight than is generally attached to it, if it is a fact, as the writer's experience would lead him to believe, that in the great majority of cases the invasion is gradual and not absolutely sudden.

The rapidity with which the blood-vessels all over the surface of the body may dilate under vaso-motor influence is illustrated by the act of blushing; and if the possibility of a local vaso-motor paresis be admitted—and there are many facts which support this view—it is conceivable that such a condition may occur in the mucous membrane of the respiratory tract. Stoerck states that he has actually observed it in the trachea during a paroxysm of asthma.

Our conclusion, therefore, is that in every case of asthma there is a nervous factor, and that this operates either upon the muscular fibres of the bronchi, causing spasm of the tubes, or through the vaso-motor system, producing hyperæmia of the bronchi, and that these conditions may be present either separately or in association.

Distension of the lungs.—This condition, which is present to an extreme degree during an asthmatic paroxysm, is, in most cases, gradual in onset and also in decline, and appears to be produced as follows:

(*a*) The bronchial obstruction induces increased inspiratory effort.

(*b*) The entering air passes the obstruction with difficulty, but the gradually increasing prolongation and force of the expiratory act shows that the air meets with still greater difficulty in escaping from the lungs.

(*c*) Expiration, although prolonged, is not sufficient to equalise the quantity of air which enters and leaves the chest; a fractional addition is therefore made to the residual air by each completed act of respiration, and in time the lungs become over-distended.

It may be objected that as the power of forced expiration is greater than that of inspiration the obstruction should be more easily overcome by the outgoing than by the incoming current of air; but it must be remembered that every individual learns to rely upon forced inspiratory efforts to remedy a defective aëration of the blood, whereas in asthma, when the lungs are distended, the condition really requires for its relief forced efforts limited to the period of expiration.

Another factor in the production of this state of extreme inspiratory distension is the compression of the smaller bronchi by the distended alveoli—an effect necessarily most felt during expiration.

There must exist in every case of asthma some condition of the nervous system which renders it possible for stimuli, unheeded in healthy individuals, to influence the vagus or sympathetic nerves, and produce the effects already described. As to the nature of this change nothing definite can be stated.

Etiology of the disease.—*Age.*—The affection may appear at any age. In 31 per cent. of the cases tabulated by Hyde Salter, the disease originated during the first ten years of life.

Sex.—All writers agree that males are more subject to the disease than females. It has been stated that the proportion is two to one.

Family and individual constitution.—In the writer's experience it is more common to find in the family history of the subjects of asthma evidence of a tendency to pulmonary disease than to nervous disorders, and in a considerable number of cases the parents or some collaterals have been the subjects of tuberculosis. Asthma may, however, occur with migraine, hysteria, and other neuroses, or more serious nervous disorders such as epilepsy. The disease may be hereditary (37 per cent. Hyde Salter) or there may be a family predisposition to it in one generation without evidence of heredity. It is perhaps more common for one member of a family to be asthmatic, another to suffer from paroxysmal sneezing, and others from hay fever.

Asthma may be associated with the fibroid form of pulmonary tuberculosis, or it may follow arrest of chronic tuberculosis, emphysema of the lungs being probably present. We have long made it a rule in all cases of asthma to examine the sputum for tubercle bacilli, as if this be omitted the true nature of many cases will be overlooked.

Course.—When the disease appears in infancy or childhood the liability to attacks may diminish as age advances and may ultimately disappear; the child is then said to have 'grown out of it.' The longer the duration of the disease, the less likelihood is there of such a favourable termination, as the secondary emphysema thereby induced tends to give permanence to the condition. The duration of the intervals between the paroxysms depends chiefly upon the absence of exposure to the possible exciting cause.

The shorter the intervals between the attacks the greater is the degree of emphysema which results; and to the emphysema, quite as much as to the disease itself, the typical appearance of the asthmatic patient is to be ascribed (*vide* under Emphysema, p. 171).

Emphysema once established tends to increase with each attack and the condition is then necessarily attended by a liability to those changes in the right side of the heart and in the venous system which are present in that disease.

Diagnosis.—In laryngeal or tracheal stenosis, the dyspnœa is of the inspiratory type, whereas in asthma it is expiratory. Laryngeal obstruction is characterised by increased respiratory movement of the larynx. This is absent in asthma, but it is also absent in tracheal stenosis. Asthma is marked by wheezing, the other affections named by stridor; in asthma the paroxysm is accompanied by extreme inspiratory distension of the lungs, an attack of dyspnœa of laryngeal origin by diminution in the size of the chest, and elevation of the diaphragm.

In some cases of acute bronchitis there may be an element of spasm in the dyspnœa, and conversely a catarrhal factor may complicate an attack of asthma. The presence of catarrh is usually well known to the subjects of asthma, with whom a favourite expression is: 'I don't mind the asthma if you can relieve the bronchitis.' It is indicated by the presence of cough, expectoration, and catarrhal sounds to an extent to which the patient is unaccustomed during an ordinary attack of his ailment.

Cardiac dyspnœa, a preferable term to cardiac asthma, is distinguished by the 'sighing' or panting character of the respiration, which is much quicker than in asthma, and lacks the characteristic prolongation of the expiratory act.

Emphysema with bronchitis may be accompanied by paroxysms of dyspnœa, indistinguishable from those of asthma by their clinical character alone. This constitutes the condition known as 'bronchial asthma.'

An attack of hay fever, in which the bronchi are involved, constitutes in our opinion one form of true asthma. The history of the case and the time of year during which the attacks occur should lead to the recognition of the exciting cause.

In uræmia the type of dyspnœa may be either expiratory or inspiratory, or there may be simply hurried breathing.

An *aneurysm* of the arch of the aorta compressing the trachea or left bronchus may give rise to paroxysms of dyspnœa of the asthmatic type, and, unless a careful examination of the patient is made, the true nature of the case may easily be mistaken. We have, however, more often seen such cases diagnosed as 'bronchitis and emphysema' than asthma. The presence of 'tracheal tugging' is a sure sign of aneurysm and is rarely absent in such cases. The brassy cough, characteristic of tracheal pressure, often excites suspicion before the examination is begun. A heaving impulse in the upper sternal region and dulness over the manubrium, with weak breathing over the left upper lobe (when the left bronchus is compressed), are signs not difficult to discover if intelligently sought for, but otherwise very easily overlooked.

Prognosis.—Death rarely occurs during a paroxysm, but Hilton Fagge relates a case in which life was only saved by the imely performance of artificial respiration. As already stated, matic children may lose the tendency to the disease by the time they reach the age of puberty, but that in our experience is the exception rather than the rule. Nevertheless, if the intervals between the attacks are long, and middle age has not been passed, there is always a possibility of recovery. Much depends upon the occupation and circumstances of the patient, and particularly upon whether, by residence in a place with a suitable climate and in other ways, he is able or has sufficient self-control to avoid the exciting causes of the attacks.

The amount of emphysema present, and the condition of the right side of the heart, are the main factors in prognosis.

Although it is possible to point to individual cases of asthma in which the disease commenced in early life, and the patient lived to advanced age, they are certainly exceptional. There can be no doubt that as, a rule, such persons do not usually 'live out their expectation,' and are rightly charged by assurance companies a higher rate of premium than healthy individuals.

Treatment.—To offer advice to a sufferer from asthma without having first obtained from him a minute account of his own observations of his case, which are often most accurate, is a sign of inexperience, which he is usually not slow to observe. A patient is not unlikely to impart more information than he receives.

Attention should first be given to the exciting causes of the attacks. When these are clear the indications are obvious. In many cases, however, the object of the patient is to find some drug which will remove the effect whilst the cause is allowed to remain—a mode of treatment which, although possibly successful for a time, is certain to fail in the end.

If, for example, a visit to a certain place induces an attack, that place must be avoided; for the longer the patient stays there the worse he becomes, and immunity will not follow either repeated visits or prolonged residence. The same rule obtains in cases in which attacks are induced by certain articles of diet; the obnoxious articles must be avoided, or they will assert their influence in spite of drugs.

Asthma is a disease of 'exceptions,' and to the above statements exceptions may be found.

As regards *locality* almost every asthmatic is a law to himself. The only general statements which can be made are that the majority of patients find that in the smoky atmosphere of a large town they are more free from attacks than elsewhere, and that a residence at the seaside is usually beneficial to those who suffer from hay asthma.

Patients in whom catarrh of the over-sensitive bronchial mucous mbrane is the usual exciting cause of a paroxysm must obviously id exposure to cold and damp, and should if possible reside in a m and dry climate. Cod-liver oil may often be taken with ntage by them all through the winter months.

ocalities in which the soil is dry and sandy, and suitable for wth of the Scotch fir, are found by experience to suit many f asthma; such are the neighbourhoods of Ascot, Aldershot, idge, and Bournemouth.

We have known boys who had been subject to attacks of asthma of moderate severity, and in whom only a slight degree of emphysema was present, lose the tendency entirely as the result of a residence at St. Moritz or elsewhere in the Engadine.

Cases in which the nervous factor is predominant, and in which catarrh and emphysema, if present, are so in a moderate degree, are most likely to derive benefit from a stay at a high altitude.

Many patients resort every year to Mont Dore, in Auvergne, for the relief of asthma, and derive great benefit from treatment there. The most important factor in the treatment is the inhalation under pressure of a hot dense vapour derived from the warm alkaline and feebly arsenical springs of the place. For a complete account of the Mont Dore treatment of asthma we must refer the reader to Dr. Burney Yeo's work on 'Climate and Health Resorts.'

Some cases of asthma dependent upon morbid conditions of the naso-pharynx are relieved by treatment at Eaux Bonnes and Eaux Chaudes.

Constipation and functional derangements of the liver are in some individuals certain precursors of an attack, and are usually best treated by the administration of mercurials and saline purgatives. Colchicum and alkaline remedies are indicated when a gouty factor is present.

Diet.—It is advisable in most cases that the principal meal should be taken at midday, as nothing is so likely to induce an attack as retiring to rest before the process of digestion is finished. In cases of so-called 'peptic asthma,' the articles of food which most often disagree are those which experience and experiment have shown to be least digestible, *e.g.* pork, hard-boiled eggs, nuts, pickles, dried and preserved meats. In our experience an attack is more often due to indigestion than to the ingestion of some special article of diet.

Sufferers from asthma should certainly be very moderate in the use of alcohol. Coffee after dinner can rarely be taken by an asthmatic patient.

In the intervals between the attacks a sufferer from asthma should endeavour to maintain his general health at as high a standard as possible, as by so doing he increases his resisting power to catarrhal affections and, generally speaking, to the exciting causes of the paroxysms. It is, however, unfortunately true that attacks sometimes occur when the patient is feeling unusually well.

When emphysema is present to a marked degree, much benefit may be derived from a course of compressed air baths, such as is described in the chapter on Emphysema (*vide* p. 178). If, however, as sometimes happens, the first trial of this treatment is followed by an attack of dyspnœa shortly after leaving the bath, it is generally better to discontinue the treatment, even should the patient be willing to continue it, for we have known an extremely severe and prolonged attack to follow the second bath.

The drug which we have found of most service in warding-off attacks is certainly iodide of potassium. It may be given in doses of five grains three times daily at first, and the quantity may be gradually increased until the patient is taking forty-five grains or a drachm in twenty-four hours. The æthereal tincture of lobelia (♏xx) or extract of stramonium (gr. $\frac{1}{4}$) may be added to it. Arsenic taken internally during the intervals between the attacks has not, in our experience, been of such service as iodide of potassium. It is, however, worthy of trial and is highly spoken of by some authorities.

Some patients never retire to rest without burning nitre paper in the room or smoking a cigarette containing stramonium or some remedy of that class. Nitre paper is made by saturating blotting-paper in a solution of nitrate of potash (gr. xxx ad ℥j). The same measures are sometimes successful in warding-off an attack in cases in which an 'aura' occurs.

Treatment of the paroxysm.—At the commencement of an attack the patient should be placed in the position which experience shows to be most suitable to his particular case. He often prefers to be in an arm-chair with rather a hard seat; if in bed, the head should be raised and comfortably supported by pillows. We have known the application to the chest of a liniment, consisting of liniment of turpentine (ʒ vj) and tincture of iodine (ʒ ij), efficacious in relieving an attack in the early stage; possibly the effect may have been due to the inhalation of the vapour as much as to cutaneous irritation.

The number and variety of the remedies available for the treatment of the developed paroxysm is the best evidence of their uncertain action.

Increasing experience of the use of morphia by hypodermic injection in cases of asthma has convinced the writer that it is a remedy of the greatest value, and that provided certain obvious precautions, not special to this disease, are observed, it may be employed without risk. We have not happened to meet with cases in which the morphia habit has been thus induced, and from inquiry we do not think they are common. It is obviously necessary to be quite certain that the dyspnœa is due to the spasmodic factor and not to an independent inflammatory catarrh of the bronchi, a point already discussed under the head of diagnosis. When the drug has not been previously employed gr. $\frac{1}{6}$ is sufficient for the first injection. This may be repeated in two hours, or increased if no effect has been produced. It is true that a certain degree of tolerance is established in time, but we have rarely known a case in which half a grain did not at any time produce a decided effect. Caffeine in doses of gr. iv every four hours is often of great service during a paroxysm.

Sedative and antispasmodic remedies are largely used in the treatment of the asthmatic paroxysm; of these stramonium and lobelia are the most efficacious.

Stramonium and belladonna enter into the composition of the majority of 'asthma cures' sold as patent medicines. Nitrites are given off when these powders are burned, and upon this their efficacy

PAGE
Cardiac pneumonia 223
Other varieties 223
Influence of complications 223

CHAPTER XVII

Diagnosis 226
From pleural effusion 226
„ lobar caseous tuberculosis 228
„ hæmorrhagic infarction 228
„ typhus fever 228
„ typhoid „ 228
„ meningitis 228
„ pulmonary œdema 229
„ collapse of lung 229
Prognosis 229
Treatment 230
General 230
Antipyretic 231
In cardiac failure 232
Venesection 233
Chloral and digitalis 233
Antitoxic treatment 234
Local applications 234

Definition.—An acute febrile disease accompanied by consolidation of the lung.

The above definition is in accordance with the view now commonly held that pneumonia is a general disease, and not merely an inflammation of the lung.

The use of any adjective, such as lobar, tends to emphasise the local aspect of the affection, and is therefore to be avoided. It would be an advantage if the term 'pneumonia,' alone and unqualified, were reserved for the disease in question.

It was to be expected that the discovery of micro-organisms in the sputa and in the lungs and other organs in pneumonia would lead many to take a step further, and maintain the theory of the microbic origin of the disease; and although its truth has not yet been proved to demonstration, there can be little doubt that in the near future this view will obtain general acceptance.

Etiology.—*Climate.*—A low and changeable temperature, associated with the prevalence of cold, dry, penetrating winds, is proved by statistics to exercise a very marked effect upon the incidence of and the mortality from pneumonia. These are the meteorological conditions most often present in this country from November to March, and it is during that period of the year that the disease is most prevalent.

Individual constitution.—The idea that pneumonia, unlike any other disease, is more prone to attack the healthy and robust than those whose resisting power has been diminished by disease, exposure, excesses, or other causes, is not now, so far as we are aware, entertained, and need not therefore be considered. A careful inquiry in the great majority of cases will show that the individual, at the time of exposure to what appears to be the immediate exciting cause of the disease, was in a condition of depressed vitality.

Predisposing causes, according to the view here adopted, can only act by lowering vital activity, and so permitting the multiplication of organisms which, if previously present, were incapable either of invasion or development.

Such a condition may arise from a variety of depressing causes, such as bodily fatigue, injury, mental depression, insufficient food, indulgence in alcohol, or the presence of other diseases, of which influenza, chronic Bright's disease, albuminuria, rheumatism, and erysipelas are the most important.

Certain persons appear to be especially prone to pneumonia, one attack following another at varying intervals, the disease recurring in the lung primarily affected more often than in its fellow. A case is indeed on record of a man who suffered from the disease on twenty-eight separate occasions.

Chill.—It is a matter of common experience that a chill may be followed in different persons by very varying effects, and that if several people are similarly exposed to an injurious influence, some escape without harm, whilst in others serious illness results. Of those affected, one may have acute rheumatism, another pleurisy, and another pneumonia. Without being able precisely to define our meaning, we are accustomed to refer this to a variation in individual resisting power. Possibly the result is determined by the existence of a local as well as a general defect. When resistance is diminished, it is conceivable that morbid influences, present but inoperative under healthy conditions, may produce their effects.

The above statements do not, it is true, explain the influence of chill as a factor in the causation of a disease believed to be of microbic origin, but they indicate the direction from which, with a fuller knowledge of the subject, light may come in the future.

Traumatic causes.—Injury to the chest may be followed by an attack of pneumonia, and the disease has been occasionally observed to follow an injury to other parts of the body.

Pythogenic causes.—The evidence that emanations from sewers and insanitary surroundings predispose to pneumonia is so complete that the possibility of a pythogenic origin of the disease must be admitted. In cases so arising it is by no means uncommon for several inmates of a house to be affected, and in such cases the existence of some condition of the kind should always be suspected and sought for.

The *epidemic prevalence* of pneumonia has been carefully studied of late years by many writers, and the subject is fully discussed in the very complete work of Sturges and Coupland.[1] The evidence that the disease may occur as an epidemic and affect a considerable number of the inhabitants of a given locality, or as an outbreak limited to the inmates of some large institution, such as a prison, is quite conclusive.

An epidemic of influenza has in some cases preceded one of pneumonia.

---

[1] *Pneumonia*, 2nd edit. 1890, p. 257.

Infection or contagion.—The possibility of this mode of transmission of the disease has naturally attracted greater attention since the belief in its microbic origin has gained ground. At present it can neither be affirmed nor denied. Much of the evidence upon which the belief rests necessarily deals with cases occurring in succession amongst members of the same family. That these cases may have been examples of epidemic prevalence of the disease, or have arisen from exposure to the same exciting cause, are possibilities which, although apparently excluded in some cases, cannot be entirely eliminated.

Bacteriology.—The first important contribution to the bacteriology of pneumonia was made by Friedländer (1882), who described capsulated diplococci as of constant occurrence in the exudation in the pulmonary alveoli. This organism he believed to be, and it was for a time accepted as, pathogenic of the disease. The name by which it is now known is Friedländer's Bacillus Pneumoniæ. It was, however, subsequently shown by Fränkel, Talamon, and Weichselbaum that the bacillus of Friedländer was only of comparatively rare occurrence in pneumonia, but that another, differing from it in morphology, virulence, and mode of growth, was present in the sputa in nearly all cases of the disease. This organism, which was first discovered by Sternberg and named the Micrococcus Pasteuri, was subsequently named the *diplococcus pneumoniæ*, but is now generally referred to as the Pneumococcus of Fränkel, or the Micrococcus Pneumoniæ Crouposæ.

Another micro-organism believed to possess pathogenic properties is Klein's *bacillus pneumoniæ*. This was first discovered in an epidemic of pneumonia of a very virulent type which occurred at Middlesborough in 1888.

In some cases of pneumonia only streptococci are found in the blood-vessels and alveoli. A brief description of these organisms will now be given.

Friedländer's bacillus pneumoniæ.—This organism has been found in the sputa, in the alveolar exudation, and the lung tissue in pneumonia. It occurs in various forms, and may be either spherical or ovoidal or rod-shaped. It is often in pairs, as a diplococcus, or may appear as small chains or rods. Whatever form the organism may assume, when present within the body and in sputa, it is seen to be surrounded by a clear hyaline capsule, but in cultivations this is absent. This capsule is due to a gelatinous softening of the cell wall, and is not distinctive of the organism.

A stab cultivation on peptone gelatine appears on the surface as an elevated white rounded mass, a similar growth extending along the needle track, giving to the whole the appearance of a round-headed nail; this, however, is not peculiar to the organism. The bacillus does not liquefy the gelatine.

Friedländer found that mice, inoculated from a cultivation by injection into the lung, developed pneumonia and pleurisy, and that the organism was reproduced in the lung, the pleural effusion, and the blood.

It has since been shown that Friedländer's bacillus may be present in normal saliva and nasal mucus, and also in the sputum in other diseases besides pneumonia.

The only positive statement which can be made as to the pathogenic nature of the bacillus is that in rabbits and mice it causes acute local inflammation and septicæmia. It is possibly pathogenic in a limited number of cases of pneumonia in the human subject.

Fränkel's pneumococcus (Micrococcus Pneumoniæ Crouposæ). This organism was discovered by Sternberg in 1880 in inoculation experiments on rabbits with healthy human saliva. In the human subject it is found in mucus from the nose and bronchi of healthy people. It is generally present in the lungs and in the rusty sputum of pneumonia. The organism usually occurs as spherical or oval cocci or as a diplococcus in the form of a dumb-bell or short chains of dumb-bells, or in straight chains of three or four, or in longer chains containing forty or fifty cocci. Individual cocci are often lanceolate, and vary much in size.

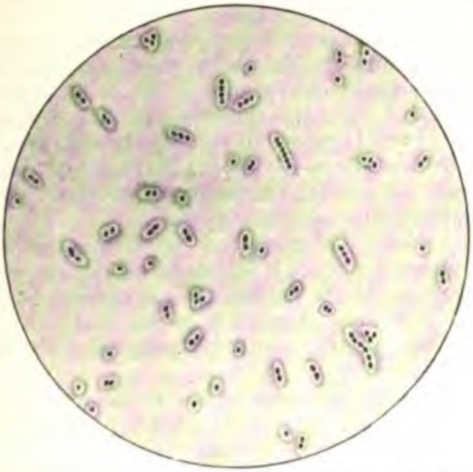


FIG. 62.—PNEUMOCOCCI IN THE SPUTUM, FROM A CASE OF PNEUMONIA

When the organisms are found in the lungs or sputa of man or in the blood of animals they are generally enclosed in a capsule, but such capsules are not found in cultivations on artificial media. Elongated cocci, resembling bacilli, are often found in the exudation of the acute peritonitis, which results from the injection into the peritoneum of the blood of an infected animal, or of virus from a broth cultivation. Pneumococci can generally be found in the recently affected parts of the lung, and in the rusty sputa in cases of pneumonia. It has also been found in the exudation of acute pleurisy, pericarditis, peritonitis, meningitis, in epidemic cerebro-spinal meningitis, and in otitis, whether occurring in association with pneumonia or independently of that disease. In some cases of ulcerative endocarditis the vegetations on the valves contain masses of this organism; it may also be present in the sputum in influenza and bronchial catarrh. Lobular pneumonia, arthritis, and suppurative lesions in other structures of the body may also be produced by the organism.

Dr. Washbourn found that when he injected mice and rabbits with cultivations of Fränkel's cocci of such a strength as to cause the death of the animals in from twelve to sixteen hours, the most marked symptoms were fever and dyspnœa. He also found that cocci were present in the blood in about three hours after inoculation, and it was crowded with them during the last two or three hours of life. When the injection was made into the peritoneal cavity peritonitis usually resulted; but pneumonia and pleurisy were very rarely found post-mortem.

G. and F. Klemperer have shown that animals may be rendered immune to the toxine of Fränkel's pneumococcus by injecting them with recent broth cultivations of the micro-organism. Dr. Washbourn repeated their experiments, and found that immunity was not conferred until twenty days after inoculation. He states that after inoculation from a virulent cultivation with a dose insufficient to kill the animal, immunity when established may last for as long a period as seventy-six days.

It has been shown that the blood serum of animals which have been thus rendered immune, if injected into rabbits, protects them from the toxic effects of the pneumococcus, and the observers above named state that the serum taken from patients convalescent from pneumonia possesses similar protective properties, but this assertion is denied by others.

Klein's bacillus pneumoniæ.—This organism appears as very numerous, 'short, oval, or rather longer rods, isolated or more frequently in pairs or in short chains.' [1]

In preparations stained with methyl blue, gentian violet, or fuchsin, the sheath of both the shorter and longer rods is slightly stained; the protoplasm is deeply coloured at its extremities. Gelatine plate cultures (at 20° C.) show, after twenty-four to forty-eight hours, colonies which appear as small grey points on the surface; these subsequently enlarge into uniform homogeneous plaques.

Subcutaneous injection into white mice of the sputum or of fluid from the lung, from cases of pneumonia in which this organism was present, or the injection of cultures of the same on artificial media, gave rise to 'intense inflammation, either in one or more lobes of both lungs, or in some cases in every part of both lungs.' 'Pleurisy and pericarditis with blood-stained effusion,' and in some cases peritonitis, followed the injection. Blood corpuscles and coagulated fibrine were found in the pulmonary alveoli of the animals inoculated.

The fact that Fränkel's pneumococcus has been found in the saliva and in the secretions from the nose and bronchi in healthy individuals, and also in the many affections already named, appears at first sight a strong argument against its acceptance as the cause of pneumonia.

---

[1] *Centralblatt. f. Bakteriologie*, Bd. v. 19, p. 626.

It may, however, be pointed out that the same micro-organism may produce different symptoms when it attacks different parts of the body. There is, for example, a wide difference between the clinical aspects of a case of acute miliary tuberculosis of the lungs, and one of tubercular disease of the peritoneum. Dr. Washbourn has shown[1] that the constitutional symptoms of pneumococcus pleurisy may be precisely similar to those of pneumonia, and our experience confirms this statement, as we have observed many cases, diagnosed as pneumonia and presenting the general symptoms of that disease, in which post-mortem there was acute inflammation of the pleura without any true pneumonic consolidation of the lung.

It is not yet clear why an acute pneumococcus infection of the pleura should produce symptoms like those of pneumonia, whilst such symptoms are absent when infection occurs elsewhere. Possibly the extent of the serous membrane and the anatomical relationship of the parts may serve to explain the fact. Dr. Washbourn suggests that in other cases of pneumococcus infection (*e.g.* meningitis) the general symptoms of pneumonia are masked by those of the local lesion.

Organisms which are normally present in the healthy subject cannot produce toxic effects unless the resisting power of the individual has for some reason been diminished to such an extent as to allow of their multiplication, and probably in all diseases of microbic origin the relation between the dose of the virus and the resisting power of the individual is the factor which determines the occurrence of the disease.

If future research should confirm the view that in pneumonia the pneumococcus of Fränkel is not invariably present, the relation of that organism to pneumonia must be different from that of other organisms, which are regarded as specific, to the diseases with which they are associated—different, for example, from that between the tubercle bacillus and tuberculosis.

For these cases the term 'non-pneumococcus pneumonia' has been suggested.

Our present knowledge of the bacteriology of pneumonia appears to justify the following statements:—

1. Pneumonia is accompanied by the growth and development of micro-organisms in the inflamed lung and in other organs and tissues of the body.
2. Neither Fränkel's pneumococcus (?), Friedländer's bacillus, nor Klein's bacillus is present in every case of pneumonia.

---

[1] 'Case of Pleurisy caused by the Pneumococcus, with Constitutional ms resembling those of Pneumonia,' by Dr. Washbourn, *Med.-Chir.* ol. 77, p. 179.

3. The organism which is most commonly met with, and which apparently possesses the most decidedly pathogenic properties, is Fränkel's pneumococcus.
4. Friedländer's bacillus pneumoniæ, Klein's bacillus pneumoniæ, the streptococcus pyogenes, the staphylococcus pyogenes aureus or albus, may also be present in pneumonia, either separately or in association with other organisms.
5. The pneumococcus is probably identical with the microbe of sputum-septicæmia or the micrococcus Pasteuri, and may be present in secretions from the mouth and nose of healthy people.
6. The pneumococcus may give rise to a variety of lesions independently of pneumonia.
7. As a result of the multiplication of the pneumococcus within the body a toxic substance is produced which gains access to the blood.
8. The phenomena attendant upon the 'crisis' of the disease are probably due to the development of an antitoxin in the blood, and by this means the action of the virus is arrested.
9. By inoculation with attenuated cultivations of the pneumococcus animals may be rendered immune to the disease.
10. The blood serum of immunised animals will protect other animals and possibly human beings from the action of the pneumococcus.
11. The blood serum of patients convalescent from pneumonia may possibly possess similar protective properties.

Morbid anatomy.—In the course of an attack of pneumonia the lung passes from its normal, spongy, crepitant condition into one of more or less complete consolidation. If recovery takes place, the lung is restored to its previous state; if death ensues, the appearance, post mortem, of the affected area varies according to the stage to which the pathological process has attained. The terms (1) engorgement, (2) red hepatisation, (3) grey hepatisation, (4) purulent infiltration and (5) resolution have long been used to describe the various stages of pneumonia.

1. *Engorgement.*—Owing to an intense congestion of the pulmonary vessels the part of the lung affected is of a deep red colour, enlarged, œdematous, pits upon pressure, and is of increased density. On section a frothy blood-stained fluid exudes, and on firm pressure of the cut surface the lung breaks down. It still, however, contains some air, and a portion placed in water either floats upon the surface or sinks to a certain depth, according to the degree of consolidation present. On microscopical examination the capillaries of the alveoli are seen to be distended with blood, and red corpuscles and a few leucocytes and large flattened epithelial cells may be found within the air vesicles. The epithelial cells lining the vesicles present a swollen and granular appearance. The vessels of the pleura covering the affected area are congested, and subpleural hæmorrhage may be present.

2. *Red hepatisation.*—The enlargement is now more marked, and the impressions of the ribs upon the surface may be obvious;

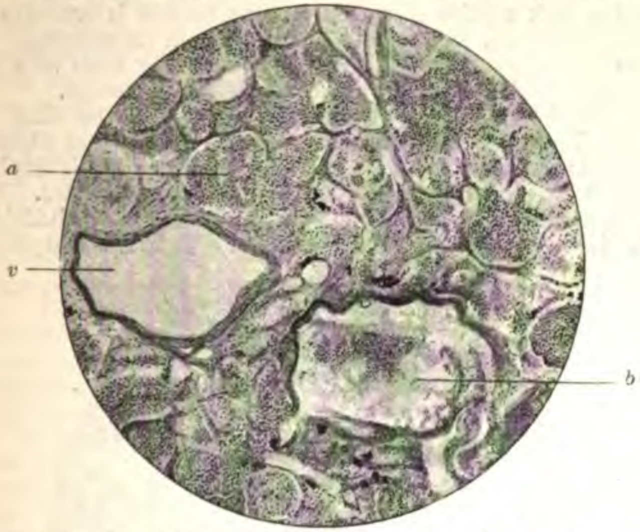
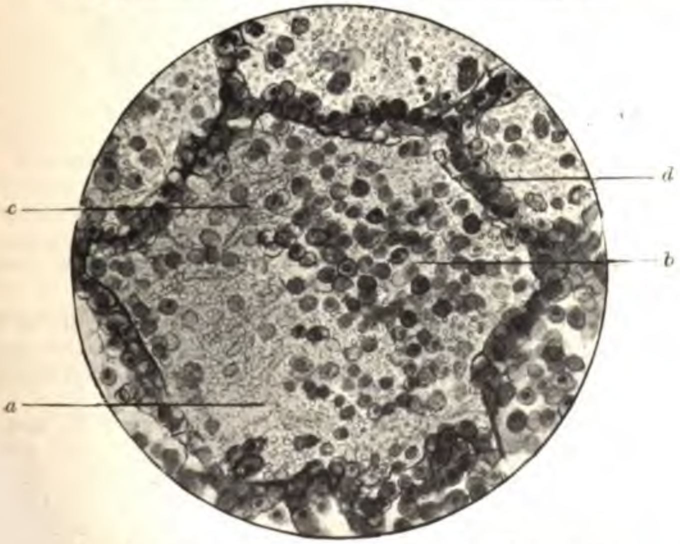


FIG. 63.—PNEUMONIA. RED HEPATISATION. LOW POWER

Showing, *a*, alveolus filled with cellular exudation; *b*, bronchus filled with fibrinous and cellular exudation similar to that in alveoli; *v*, vessel containing blood



., 64.—PNEUMONIA. RED HEPATISATION. HIGH POWER

filled with (*a*) red blood corpuscles, (*b*) leucocytes and proliferated cells, (*c*) fibrin; (*d*) the thickened alveolar wall consolidation is complete, the part sinks in water, does not crepitate, and on firm pressure breaks with a granular fracture. On section it is of a dark reddish-brown colour, the surface is granular and no longer glistens, and yields a rusty red fluid on scraping. The section may show that the smaller bronchi are filled with solid cylinders of fibrin, which may be drawn out with forceps.

Under the microscope the alveoli are seen to be filled with coagulated fibrin forming a fine network, the filaments of which are attached to the alveolar wall. In the interstices of this fibrinous exudation are numerous red corpuscles and fewer leucocytes. Some larger nucleated cells derived by proliferation of the epithelial cells of the alveoli are also present, more often near to the margin than in the centre of the alveolus. The vessels of the pleura and of the interlobular septa are engorged with blood, the connective tissue structures are thickened from œdema, and the bronchial mucous membrane is injected.

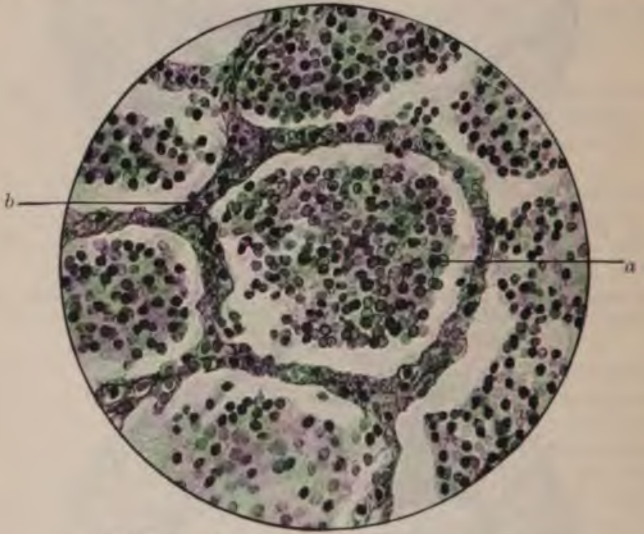


FIG. 65.—PNEUMONIA. GREY HEPATISATION

Showing, *a*, alveolus containing cellular exudation which has shrunken from the alveolar wall; *b*, alveolar wall thickened and infiltrated. NOTE.—The fibrin and red blood corpuscles can no longer be seen

The pleura, particularly that part corresponding to the area of consolidation, including the interlobar septa, may be covered with a thick layer of lymph, or the pleural cavity may contain a varying quantity of clear or turbid, sometimes blood-stained, fluid or pus. Flakes or larger masses of coagulated lymph may be present in the fluid, and it generally contains pneumococci.

3. *Grey hepatisation.*—Between grey and red hepatisation there are numerous intermediate conditions to which the terms reddish-

grey or greyish-red are applied, according as one or the other colour predominates. The appearances they present have been likened to those of red or grey granite.

This change of colour is probably due to various causes: to increasing exudation of leucocytes, to emptying of the vessels of the alveolar wall by pressure, and to absorption of the red corpuscles by leucocytes. The grey colour is to some extent a post-mortem change.

In a case of complete grey hepatisation the lung is even larger than in the last stage, and may weigh from three to four pounds or even more. The section is of a grey or greyish-yellow colour.

The granular character of the surface is now less marked. If much œdema is present the surface may even have a shiny glistening appearance. The consolidated part is less firm than in the red stage. The fluid which exudes on pressure or scraping is milky or yellow and almost purulent. The bronchi, even those of a large size, and in some cases the main bronchus of the lobe, or even that of the affected lung, may be filled with solid cylinders of fibrin (massive pneumonia). On microscopical examination the exudation now no longer completely fills the air vesicles, the fibrin and red corpuscles have disappeared and have been wholly or in part replaced by leucocytes, which form opaque masses within the alveoli. Some of the leucocytes are seen to contain three or four nuclei, and many are undergoing fatty degeneration. The alveolar walls are also infiltrated with leucocytes to a moderate degree; but the blood-vessels, instead of being distended, are more or less empty.

4. *Purulent infiltration.*—This is merely an advanced stage of grey hepatisation, from which it does not differ in its essential pathological features. The lung is softer, it breaks down more easily, and is more distinctly yellow in colour. The section has entirely lost its granular appearance, and the fluid exuding on pressure is, like the contents of the alveoli, more decidedly purulent.

Terminations of pneumonia.—1. *Resolution.* During this stage the cell elements of the exudation undergo fatty degeneration and break down, and, like its other constituents, are converted into a liquid capable of being absorbed by the lymphatics, by the agency of which it is in great part removed. In many cases the lung is restored to its spongy condition by this process of absorption, without any appreciable assistance from expectoration.

2. *Abscess.*—The number of cases of pneumonia terminating in the formation of an abscess is extremely small. The process, when it occurs, appears to be as follows: owing to the vitality very acute character of the inflammatory process, and to the diminished supply of blood
e part, the vitality of a considerable area of the affected lung
reduced that it ultimately undergoes necrosis and breaks down.
faction of the necrosed area occurs, but is usually incomplete,
rtions of the dead tissue remain as solid masses and as such
expectorated along with large quantities of pus. A cavity
ged walls occupies the site of the portion of lung which has
troyed.

3. *Gangrene.*—This is also a rare termination of the disease, as is shown by the fact that it was met with in only two out of 128 fatal cases of pneumonia occurring at the Middlesex Hospital during ten years. Both patients were males æt. 32 and 33 respectively.

The process is the same as the last, but so acute that large masses of the lung undergo necrosis, and putrefactive decomposition of the necrosed tissue occurs.

Lobar distribution of pneumonia.—It has long been recognised that the disease more frequently affects the right lung than the left. This is true of both sexes and at all ages.

In 108 of the 128 fatal cases above referred to, one lung only was affected; the right was the site of the disease in 74, and the left in 34. The proportion of 7 to 3 agrees fairly with the results obtained by other observers. In 64 fatal cases in which one lobe only was consolidated the relative frequency was as follows:

Right lung—	
Upper lobe	9
Middle „	—
Lower „	24
Left lung—	
Upper lobe	11
Lower „	20

Evidence derived solely from post-mortem records is not of course conclusive, but these figures hardly bear out the generally received view that the right lower lobe is far more often the site of the disease than the left, the different frequency of affection of the two, when only a single lobe is involved, not being very great. If, however, the whole 128 cases are included, the greater frequency of involvement of the right lower lobe is seen to be very marked, it having been consolidated in 64 cases as compared with 39 in which the left lower lobe was involved. This experience is in accord with that of Wilson Fox,[1] who states that the generally accepted view 'is not true of the lower lobes alone.'

The lobar distribution of the disease in the whole 128 fatal cases was as follows:

Right lung—		
Upper lobe consolidated	58	times
Middle „ „	44	„
Lower „ „	64	„
Left lung—		
Upper lobe consolidated	28	times
Lower „ „	39	„

All the lobes of the right lung were affected in 21 cases, and of the left in 9 cases. One lung only was affected in 108 cases, both lungs in 20.

An analysis of these cases appears to show that (*a*) pneumonia far more often attacks one lung than both; (*b*) the right lung is more often affected than the left (7 : 3); (*c*) when the disease is limited to one lobe, the lower lobes are more often affected than the upper; (*d*) the middle lobe of the right lung is rarely affected except in association with the other lobes of that lung.

---

[1] [illegible]

These statements are in accord with those of other observers, and with both clinical and pathological experience.

State of the blood. Leucocytosis.—The accompanying table, taken from the Report for 1895 of the medical registrar of the Middlesex Hospital (Dr. Wethered), shows the degree of leucocytosis present in six consecutive cases of the disease.

Cases	Date	Temp.	Red corpuscles	White	Result
Male, æt. 22	Feb. 7	104·8°	4,900,000	1 to 120 red	Recovery
1 Right	,, 8	99·2°	4,900,000	1 ,, 300 ,,	
2 Left	,, 11	104·4°	3,800,000	1 ,, 300 ,,	
Male, æt. 23	Feb. 14	103·6°	3,200,000	1 to 100 red	Recovery
Right apex	,, 16	98·6°	4,530,000	1 ,, 450 ,,	
Female, æt. 29	Mar. 30	102·0°	3,200,000	1 to 280 red	Recovery
Left base	Apr. 3	98·0°	3,200,000	1 ,, 450 ,,	
Male, æt. 20	Apr. 27	102·8°	—	1 to 250 red	Recovery
Left apex	May 2	98·0°	—	1 ,, 400 ,,	
Male, æt. 23	May 13				
Double	3rd day after admission	101·8°	—	1 to 117 red	Death
Male, æt. 19	May 24	103·4°	4,950,000	1 to 240 red	Recovery
Right apex	,, 27	98·4°	5,000,000	1 ,, 420 ,,	

Cabot's conclusions on the subject of leucocytosis in pneumonia are as follows:

(1) At the onset of the disease, or at any rate within a few hours, leucocytosis is present, and persists throughout the febrile period.

(2) At the time of a true crisis, and often a few hours previously, the number of white corpuscles begins to diminish. The fall in number is gradual, and the pyrexia disappears before the leucocytosis.

(3) If resolution is delayed, leucocytosis continues and gradually diminishes in degree as the temperature falls.

(4) The factors which chiefly influence the degree of leucocytosis are the severity of the inflammation and the resisting power of the individual.

  (*a*) If the dose of the poison is small and the reaction vigorous, the degree of leucocytosis is slight.

  (*b*) If the dose is large or moderate and reaction vigorous, there is marked leucocytosis.

  (*c*) If the inflammatory changes are marked and reaction is feeble, there is no leucocytosis.

The indications for prognosis derived from the examination of the blood in pneumonia appear to be as follows:

(*a*) The absence of leucocytosis is a very unfavourable condition. In the majority of such cases death ensues.

(*b*) The presence of leucocytosis is neither of favourable nor unfavourable import.

The indications as regards diagnosis may be stated thus:

(*a*) If there is well-marked leucocytosis, the case is probably one of pneumonia, as typhoid fever, malaria, and influenza may from this fact be excluded.

(*b*) Leucocytosis is present in both pneumonia and capillary bronchitis.

(*c*) In old and very young subjects leucocytosis may be absent.

The leucocytosis in this as in other pyrexial affections is to be regarded as a defensive process on the part of the organism against the virus of the disease.

Complications of pneumonia.—

Frequency of Complications and of Associated Lesions in 128 *Fatal Cases of Pneumonia*

			Frequency per cent. in fatal cases
Pleurisy with effusion		32	25
Right pleura	11		
Left ,,	14		
Both pleuræ	7		
Empyema		3	2·3
Pericarditis		17	13·2
Endocarditis, acute		5	3·8
,, chronic		22	17·1
Pulmonary tuberculosis in arrest		21	16·4
Right lung	3		
Left ,,	8		
Both lungs	10		
Emphysema		18	14
Acute bronchitis		12	9·3
Pulmonary œdema		65	50·7
Lobar collapse		9	7
Pulmonary gangrene		2	1·5
Mediastinitis		4	3·0
Nephritis, acute		1	0·7
,, chronic		19	14·8
Cirrhosis of liver		6	4·6
Meningitis		2	1·5

The above table affords a rough, but probably an accurate, view of the relative frequency of the more important complications and associated pathological lesions met with in fatal cases of pneumonia, but gives no idea of the frequency with which complications are met with in cases that recover.

Pleurisy.—Some degree of inflammation of the pleura occurs in nearly all cases of pneumonia, and this commonly leads to an exudation of lymph upon the surface of the membrane. It will be observed, on reference to the preceding table, that in a large proportion of fatal cases fluid also is present. This may be sero-fibrinous, hæmorrhagic, or purulent. In a case recently under our observation, a double pneumonia was complicated by a hæmorrhagic effusion on the right side and an empyema on the left. The former was aspirated and the latter drained, and recovery followed.

Pleural effusion in children has, as is well known, a greater tendency to become purulent than in adults. This is true also when the effusion is a complication of pneumonia. The pleural exudation in pneumonia generally contains pneumococci.

Bronchitis.—Some degree of injection of the mucous membrane of the bronchi is almost always present. When the change is more advanced than this, the capillary form of the affection is the most common, the smaller tubes in many cases being filled with puriform fluid. In very severe cases the bronchi, from the smaller tubes upwards, as far as the main divisions, may be found filled with solid cylinders of fibrin, and in nearly all cases a similar exudation can be demonstrated in the smaller tubes of the affected area.

Œdema.—This may occur either in the lobe nearest to the one consolidated, or in the opposite lung. It was present in one or other, or both sites, in more than half the fatal cases analysed.

Collapse of a whole lobe, although by no means rare, is far less often observed than œdema; it has a similar distribution.

Pulmonary gangrene.—Two examples of this complication were observed. Both were cases of complete consolidation of the right lung; in one the upper, and in the other the lower, lobe was gangrenous. The subject of gangrene in connection with pneumonia is fully discussed elsewhere (*vide* p. 222 and p. 247).

Pericarditis.—It is generally stated that this complication is most frequently met with in cases in which a portion of the left lung in contact with the pericardium is consolidated. In the fatal cases analysed, however, the pneumonia affected the right lung in ten cases, the left in six, and both lungs in one case. The extent of the lesion varies. There may be only a few flakes of lymph upon the surface of the membrane, or the sac may be distended by half a pint or more of purulent fluid. Pneumococci are usually present in the exudation.

Acute endocarditis.—The ulcerative form of endocarditis was found by Dr. Osler in 11 per cent. of fatal cases, and other observers have confirmed his observations of its occurrence in association with pneumonia, and also of the fact that pneumococci are present in the vegetations. In the five fatal cases contained in the table given above, in which acute endocarditis was present, recent vegetations were found, but none of the cases were of the ulcerative type.

Chronic endocarditis, whether of the mitral or aortic valves, is found in a considerable proportion of fatal cases of pneumonia (17 per cent.), and is met with most frequently in those in which the right lung is alone affected.

Thrombosis of the branches of the pulmonary artery in the consolidated area is not infrequently observed in fatal cases, and the whole of the vessel in the affected lung has been found to be

On any day from the third to the tenth, and most commonly on the seventh or fifth, from the date of the rigor, the patient passes through what is termed a 'crisis.'

Phenomena of the crisis.—The first sign of the onset of this favourable change is in many cases an alteration in the facial expression from anxiety to calm; delirium and restlessness disappear, the cheeks lose their flush, and the face becomes pallid. A tendency to sleep is now observed. This is at first broken, but gradually deepens, and may last for twelve hours or more. The skin, previously hot and dry, is now moist, perspiration follows, and becomes profuse. The temperature falls in the course of twelve or eighteen hours from perhaps 104° to 98° or 97°, but may rise again to 99° or 100° on the following day, then falling again to the normal or below it, and so remaining during convalescence. The respiration becomes markedly slower, and dyspnœa, if it has been present, disappears. The pulse falls in frequency, possibly to 60 or even less. The cough becomes looser, but the expectoration retains its rusty character, although in a less degree.

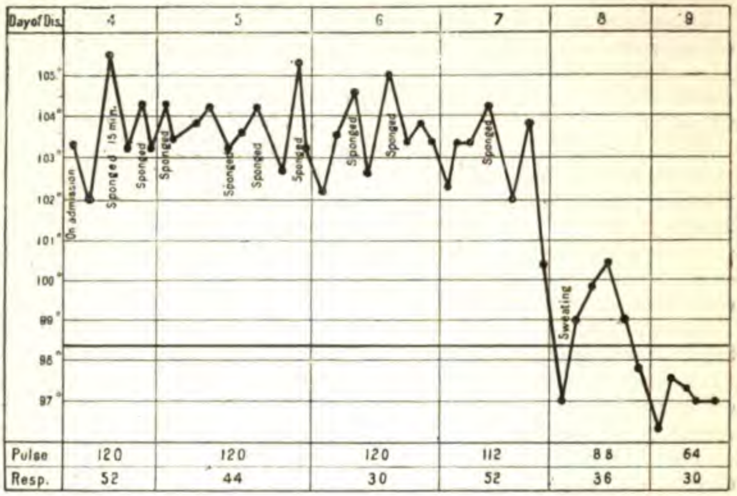


FIG. 66.—PYREXIA IN PNEUMONIA. CRISIS ON SEVENTH DAY OF THE DISEASE

On waking from his prolonged sleep a change is observed in the general condition of the patient such as rarely occurs in so short a time in any other disease. Not infrequently he says that he feels 'quite well, but rather weak.' In cases which have been accompanied by marked delirium, however, the mind may not become quite clear for some days. Appetite now returns. Urine is passed in larger quantity, the chlorides reappear, and in a few days the quantity of urea and uric acid falls to normal.

Convalescence is, as a rule, short and free from complications, recovery being generally complete in about three weeks.

Such is the course of an attack of the disease of ordinary severity, but, like most other diseases, it is subject to great variations. This section of the subject is considered in the following chapter.

Physical signs.—*The stage of engorgement.*—The phenomena of the disease may be present for three or even four days without any signs being discoverable by physical examination. This, however, is quite the exception; a definite diagnosis can, as a rule, be made by the end of the second day.

It is usually stated that the earliest sign is weakness of the breath sound over the affected area, and this is in accordance with our experience; but some writers have said that it is harsh at this period. Crepitation next becomes audible, chiefly at the end of inspiration.

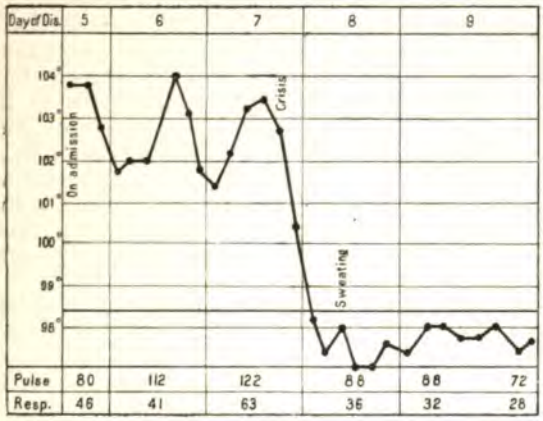


FIG. 67.—TEMPERATURE IN PNEUMONIA. CRISIS ON SEVENTH DAY OF THE DISEASE

As the exudation continues to be poured into the alveoli and the lung loses its spongy character, the resonance of the percussion note is gradually impaired, until finally it is replaced by dulness when consolidation is complete.

A pleuritic friction sound may be present at this stage of the disease.

Stage of hepatisation.—The characteristic sign of this stage is the tubular quality of the breath sound. Crepitation may no longer be audible, but in the majority of cases both signs are at first present together, crepitation subsequently disappearing whilst the breathing retains its tubular quality. The vocal fremitus is generally increased and bronchophony is present, but a layer of coagulated lymph separating the pleural surfaces may diminish the former, and give to the voice sound a distant and somewhat bleating character. The percussion note over the upper lobe in a case of basic pneumonia may now present the Skodaic quality. The heart sounds are often well conducted through the consolidated lung.

Stage of resolution.—Either before or at the time of the occurrence of the crisis a change takes place in the physical signs. This in many cases consists in a lessening, but never in the sudden disappearance of the tubular quality of the breath sound. It gradually loses its 'whiffing' and often almost metallic character, becoming first bronchial and then merely harsh. In some cases, however, whilst resolution is in progress, it may acquire an almost cavernous quality. In favourable cases the process of liquefaction of the exudation, previous to its absorption by the lymphatics of the lungs, now commences. Air again passes through the exudation, giving rise to the sound known as *redux crepitation*—a large, coarse râle of bubbling rather than crackling quality, audible both during inspiration and expiration. In favourable cases, as the process of absorption of the exudation continues, the percussion note becomes gradually more resonant, the adventitious sounds diminish, air enters more freely, and finally the lung returns to the normal condition. It is by no means uncommon, however, for the lung to remain solid for a considerable period after the crisis, and redux crepitation may not be audible at any period. Dulness at the base may continue whilst resolution is in progress, owing to the presence of a thick layer of lymph on the surface of the pleura. This exudation is ultimately in part absorbed and in part organised into adhesions.

Variations in physical signs.—Crepitation may be absent throughout the attack: this is more often the case in children than in adults. When the disease supervenes upon bronchitis, rhonchi and bubbling râles may be present in addition to crepitation, and heard during the stage of consolidation and in that of resolution.

A tympanitic or a tubular percussion note may be elicited in some cases, the latter condition probably indicating either that consolidation has not extended to the surface of the lung, or that areas of consolidation and of tissue containing air are intermingled. When pleuritic effusion is present in addition to consolidation of the lower lobe, the percussion note anteriorly may be of the most marked Skodaic character, and the breath sounds over the dull area of a character which suggests the presence of a cavity.

In the early stage intense pain in the side may be unaccompanied by pleural friction. This arises in some cases from the fact that the diaphragmatic surface of the pleura is chiefly involved. Auscultatory signs may be conducted from one side to the other—a condition, however, not peculiar to pneumonia. This is most often present at the bases, and may give rise to a diagnosis of double pneumonia when one lung only is affected. This error may usually be avoided by close attention to the character of the percussion note.

J. K. F.

CHAPTER XVI

PNEUMONIA—*continued*

VARIATIONS IN THE CLINICAL ASPECT OF THE DISEASE

We have in the preceding chapter endeavoured to present a picture of the symptoms and physical signs which characterise an attack of pneumonia uncomplicated by the presence of any of the numerous factors which may change its favourable course. These will now be described under the following headings:—

1. Onset.
2. Expectoration.
3. The crisis.
4. Pyrexia.
5. Gastro-intestinal symptoms.
6. Terminations.
7. The influence of 'type.'
    - (*a*) Latent form.
    - (*b*) Migratory form.
    - (*c*) Intermittent form.
    - (*d*) Cardiac form.
    - (*e*) Other forms.
8. The influence of complications.
    - (*a*) Bronchitis.
    - (*b*) Emphysema.
    - (*c*) Pleuritic effusion.
    - (*d*) Pericarditis.
    - (*e*) Myocardial changes.
    - (*f*) Endocarditis.
    - (*g*) Delirium tremens.
    - (*h*) Meningitis.
    - (*i*) Chronic nephritis.
    - (*k*) Parotitis.

1. *Onset.*—Marked prostration, and the early occurrence of extreme restlessness or active delirium, are, as a rule, indications that the attack will be severe. Delirium may, however, be merely the sign of an unstable nervous system. It is very often met with in alcoholic or debilitated subjects, and is then of very unfavourable import. It may in such patients appear later, about the time of crisis. Maniacal delirium, delirium tremens, and mental disorder may also in rare cases be present at the onset of an attack, which may nevertheless run a mild and favourable course.

Delirium is generally more marked in pneumonia of the upper than the lower lobes.

2. *Expectoration.*—The sputa may, instead of having the typical 'rusty' appearance, be of a bright red tint from the presence of a larger quantity of blood. This condition is more often observed in old people (hæmorrhagic pneumonia).

In a very acute case, recently observed, the patient expectorated daily for five days nearly a pint of frothy muco-purulent sputum containing myriads of pneumococci. On the sixth day it diminished in quantity and was slightly 'rusty' in character. A crisis occurred on the seventh day and was followed by recovery.

Watery sputa of a dark purple colour, to which the term 'prune juice' is applied, are usually an indication of a severe type of the disease. The expectoration may present a greenish tint, suggestive of an admixture of bile; but the colour is not due to bile pigment.

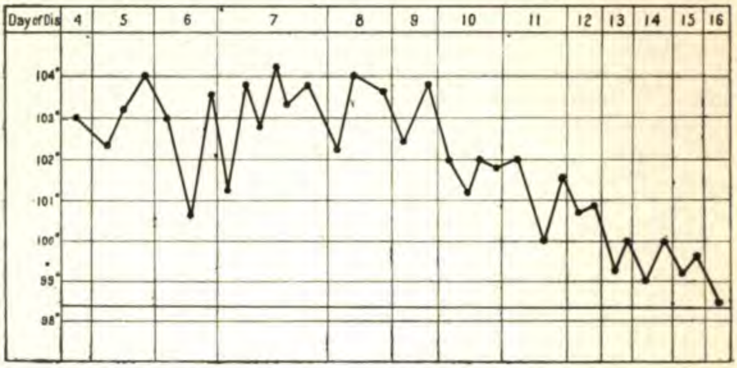


FIG. 68.—TEMPERATURE CHART FROM A CASE OF PNEUMONIA, SHOWING FALL BY LYSIS

This green sputum is a rare condition; we lately observed it in a fatal case, in which the lung remained solid for many weeks.

When pneumonia is secondary to bronchitis or influenza, the sputa may be mucoid and frothy, the rusty character being absent.

The onset of grey hepatisation and breaking down of the lung may be indicated by the sputa becoming purulent and by the presence therein of elastic tissue; should gangrene supervene, the expectoration will present the odour and appearance characteristic of that condition.

Many children and some adults pass through the various stages of the disease without expectorating.

3. *The crisis.*—The crisis occurs most frequently (22 per cent.) on the seventh day from the initial rigor, but in 15 or 16 per cent. on the fifth day, in 12 per cent. on the sixth and eighth days, and in 10 per cent. on the ninth day.

It appears that, in about 74 per cent. of all cases, the crisis occurs between the fifth and ninth days. In some tables the fourth day is credited with a rather higher percentage than the ninth.

A crisis may occur, but the pyrexia may not immediately disappear. The temperature falls, but not to normal; it subsequently rises again, but not to its previous height, and defervescence occurs by 'lysis.'

4. *Pyrexia.*—Great variations are met with in the course of the pyrexia, as evidenced by the appended charts, which are taken from cases observed in the Middlesex Hospital.

Chart of M. B.—Fig. 68 is from a woman aged 43, admitted on the fourth day with pneumonia of the right lower lobe. It illustrates the disappearance of the pyrexia by 'lysis.'

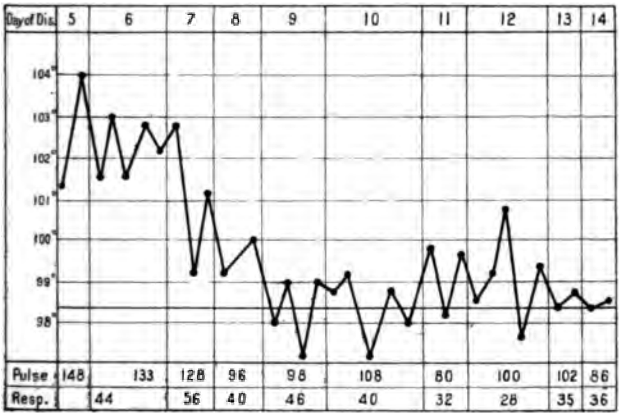


FIG. 69.—TEMPERATURE CHART FROM A CASE OF PNEUMONIA, SHOWING FALL BY LYSIS AND SUBSEQUENT RECURRENCE OF PYREXIA

Chart of J. J.—Fig. 69 is from a man aged 23, admitted on the fifth day with pneumonia of the whole of the left lung. The fall of temperature on the seventh day was accompanied by sweating, but delirium continued. After an increase of pyrexia on the following day there was a gradual decline, the temperature becoming normal on the fourteenth day, but the pulse and respirations remained frequent. Resolution was delayed and was not complete until six weeks from the onset of the disease.

Chart of J. D.—Fig. 70 is from a patient admitted on the second day. The pneumonia was of the septic type. Fränkel's pneumococci were present in the sputum, but not in large numbers. There was incomplete consolidation of almost the whole of the left lung. Resolution was not complete until eight weeks from the onset of the attack. The 'hectic' type of the temperature is very marked.

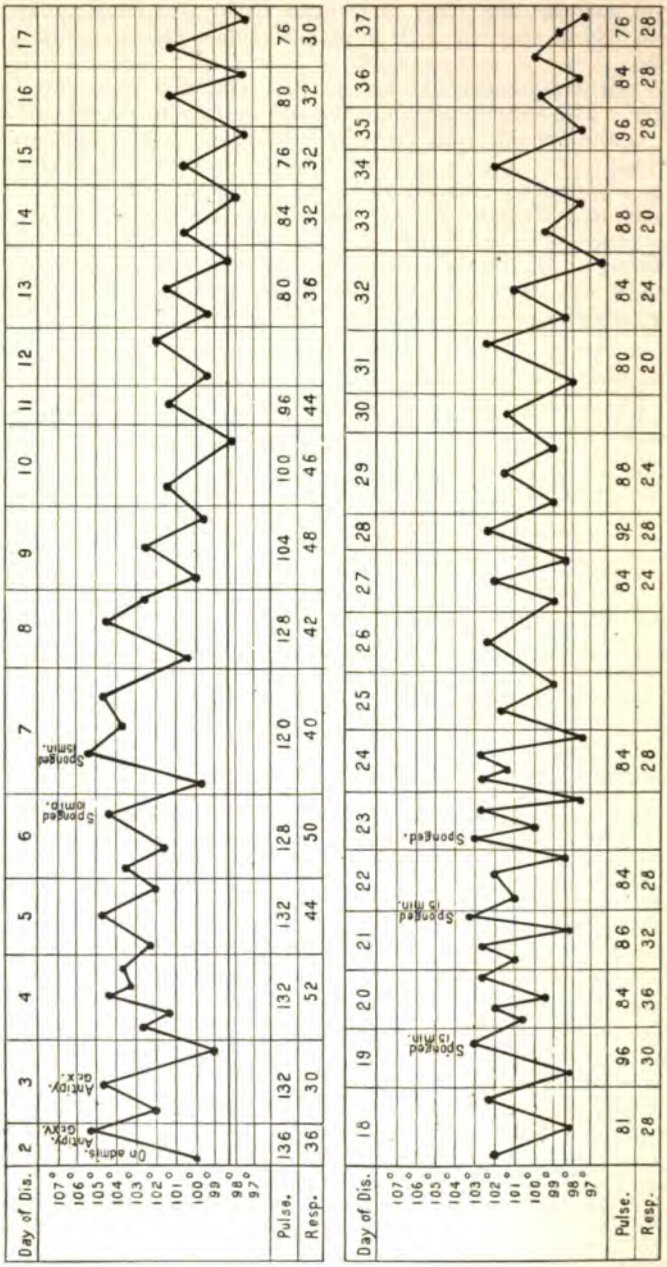


Fig. 70.—Temperature Chart from a case of Pneumonia of prolonged duration, showing 'Hectic' Temperature

rt of N. W. (Fig. 71).—The patient was a girl aged
n the fourth day with incomplete consolidation of t
be. A sudden fall in the temperature to 96·6 with

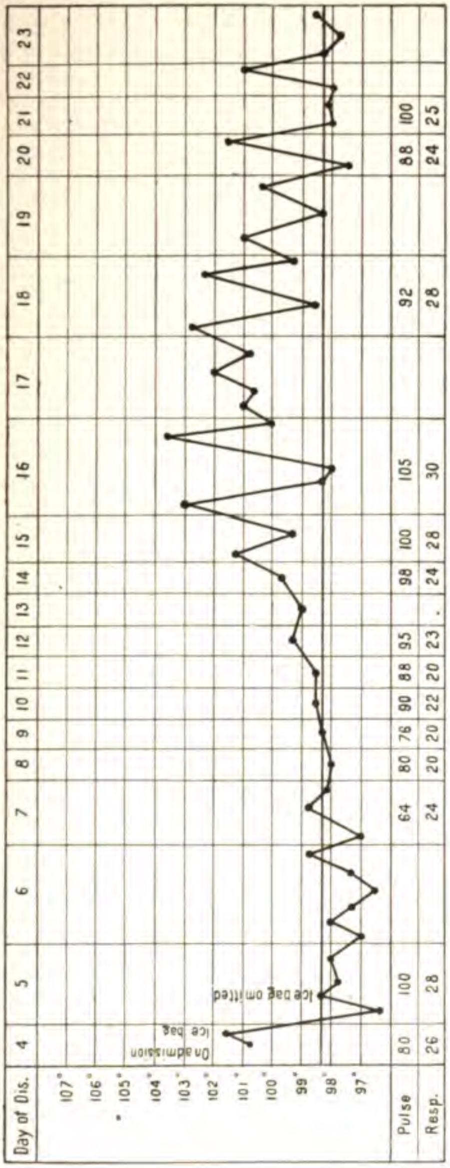


FIG. 71.—TEMPERATURE CHART FROM A CASE OF PNEUMONIA. SUDDEN FALL OF TEMPERATURE FOLLOWING THE APPLICATION OF AN ICE-BAG. SUBSEQUENT RECURRENCE OF FEVER

risis followed the application of an ice-bag to the chest. perature remained normal for five days; a relapse then with fresh invasion of the anterior part of the left lower

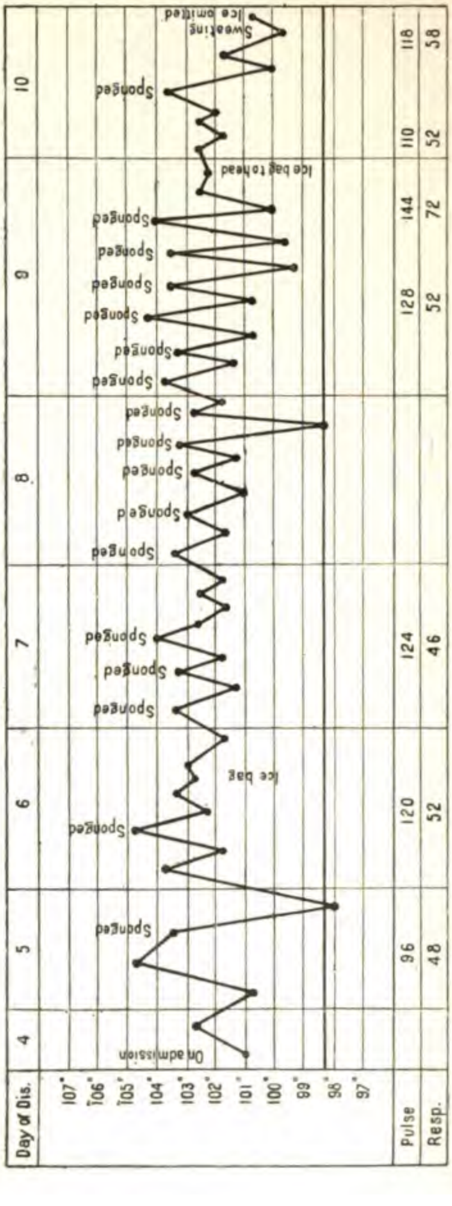


FIG. 72.—TEMPERATURE CHART FROM A CASE OF PNEUMONIA FATAL ON THE TWELFTH DAY

lobe. A period of pyrexia of a markedly intermittent character then ensued, and continued for eleven days. Resolution was not complete until twenty-seven days after the onset of the relapse.

Chart of A. A.—Fig. 72 is from a severe and fatal case of double pneumonia in a youth aged 17. The patient was admitted on the fourth day of the disease with consolidation of the whole of the left lower lobe and incomplete consolidation of the right lower lobe. Death occurred on the twelfth day.

Chart of B. F.—Fig. 73 is from a man aged 55, admitted on the second day. Complete consolidation of the right lower lobe developed, and death occurred on the seventh day. It will be observed that the temperature only once reached 104° and ranged between 101° and 103°.

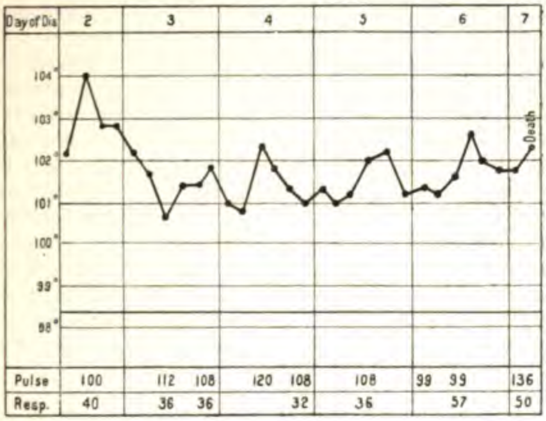


FIG. 73.—TEMPERATURE CHART FROM A CASE OF PNEUMONIA FATAL ON THE SEVENTH DAY, SHOWING MODERATE DEGREE OF PYREXIA

This is to be regarded as an illustration of the fact that pyrexia, within certain limits, is an evidence of vital activity and that a low temperature may be a sign of vital depression, such as occurs when pneumonia attacks old people or individuals weakened by starvation or chronic alcoholism. It is chiefly in such cases that death occurs although the temperature remains low.

5. *Gastro-intestinal symptoms.*—Vomiting is of frequent occurrence at the onset in children. Diarrhœa more often appears about the time of crisis, and may be present in unfavourable cases in the absence of crisis. In a case recently under our care, a severe rigor was immediately followed by violent diarrhœa, the motions for two days containing considerable quantities of blood. The temperature fell to normal on the seventh day and the patient recovered.

6. *Terminations.*—Such cases as do not run a favourable course may prove fatal during the acute period of the disease or after the

crisis. In the former case, the most common cause of death is cardiac failure. This leads to congestion and œdema of the previously unaffected parts of the lungs, as evidenced by the presence of fine crackling râles. Expectoration ceases, mucus collects in the trachea and larger bronchi, giving rise to coarse râles. The respirations become increasingly frequent, the pulse extremely rapid and feeble, the face livid, a profuse sweat breaks out, and the extremities become cold, death being preceded by a condition of semi-coma, or consciousness may be retained almost to the end. In children a convulsion may then occur.

Death may take place at the period of crisis from extreme prostration followed by collapse, but this is an event not often observed.

In rare cases, most often met with in children, after an incomplete crisis the fever may continue at a lower range for some weeks, death being ultimately due to extreme emaciation.

The febrile period may terminate in a well-marked crisis, but the lung may remain solid and subsequently pyrexia may return, and the disease ultimately prove fatal. An example of this recently came under our notice in the case of a patient who was attacked by pneumonia in Ceylon. Contrary to advice he came home before the lung had undergone resolution, although fever had disappeared. On the voyage the temperature again rose, and on his arrival was found to range between 101° and 102°. This was steadily maintained for some weeks, but the lung showed no signs of breaking down. Death was preceded by severe diarrhœa of a dysenteric character. No post-mortem examination was made. On the occasion of the writer's visit there were no signs suggesting the presence of an hepatic abscess, and none developed at a later period.

The consolidated lung may break down and an *abscess* may form, its contents being expectorated. This event is extremely rare in cases not of pyæmic origin. It is indicated by the occurrence of profuse purulent expectoration, which may be fœtid, and contain elastic tissue. The physical signs are those of softening and cavity formation. Pyrexia continues, the fever being of the hectic type; there is marked emaciation, and death may be due to septic poisoning or rupture of the abscess into the pleura and pyopneumothorax. If the abscess can be localised, such cases may be successfully treated by making an external opening, and if no operation is possible, they may after many weeks' duration terminate favourably.

Gangrene is also a very rare mode of termination, but somewhat less so than abscess formation. It is more common in apex than basic pneumonia, and is more often of the circumscribed than of the diffuse variety. Its occurrence is marked by symptoms and signs very similar to those of abscess, a gangrenous odour of the breath and expectoration being the most distinctive. Abscess and gangrene are apt to cause infection of the opposite lung from the inhalation of foul expectoration.

The subject of pneumonia terminating in pulmonary fibrosis is discussed in the chapter on Subacute Indurative Pneumonia (*vide* p. 236).

7. **The influence of type.**—*Variation in type.*—Variation in type is a characteristic of disease. To this rule pneumonia is not an exception, but of the many varieties of the affection which have been described a few only present features sufficiently well defined to be easily recognised.

(*a*) *Latent pneumonia.*—A certain amount of vital activity of the organism is necessary in order to produce the symptoms of any acute disease. If the vital activity is depressed, morbid changes may proceed and death may ensue without the manifestations of the ordinary signs of the disease. The onset and course of pneumonia in old people and in individuals weakened by starvation or alcoholic excess, or the subjects of chronic renal disease, are often extremely insidious, and sometimes so slightly marked by symptoms that the true nature of the case is only discovered on post-mortem examination.

(*b*) *Migratory pneumonia.*—This term is applied to cases of pneumonia characterised by 'successive invasion of different parts of the lung, while the physical signs disappear from the parts first attacked.' The term does not merely imply that the disease spreads from one part of the lung to another, or that a relapse occurs after apparent recovery, but that the extension into one part coincides with resolution in another, and it is this which constitutes the distinguishing characteristic of the affection.

Each successive invasion may be marked by a rigor with a repetition of many of the features of the onset of the disease, the course of which may thus be prolonged for several weeks. Such cases are rarely observed. The prognosis is not so unfavourable as would on *à priori* grounds appear to be likely.

(*c*) *Intermittent pneumonia.*—The co-existence of malaria and pneumonia may give to the pyrexia an intermittent type, but a similar course of the fever has been noted in cases where there was no reason to suspect the presence of malaria. The period of apyrexia is said to vary from twelve to thirty-six hours, during which no change is observed in the physical signs. We are unable to describe such cases from our own experience of the disease; they are certainly not common in this country.

(*d*) *Cardiac pneumonia* is a term applied to the disease when it occurs in the subjects of chronic affections of the mitral valve, especially stenosis. The peculiar features it presents are scarcely sufficient to justify a separate description. They are said to be the absence of pleurisy and the long continuance in the stage of red hepatisation.

(*e*) The varieties of the disease, to which the names *typhoid*, *bilious*, *puerperal*, *recurrent*, *pythogenic*, and *cerebral* have been applied, are, in our opinion, either not sufficiently well defined or not so commonly met with in this country as to require detailed consideration.

8. **The influence of complications.**—Of the conditions

antecedent or subsequent to the onset of the attack which may modify its course, the more important are bronchitis, emphysema, pleuritic effusion and empyema, pericarditis, endocarditis and myocarditis, meningitis, chronic renal disease, and parotitis.

Complications, although of rare occurrence in pneumonia, are of grave import, the mortality of cases in which they occur being nearly four times greater than in uncomplicated cases (Huss).

(1) *Bronchitis*.—When widely diffused this is a serious complication, as it causes marked dyspnœa, and tends to increase the strain upon the right cavities of the heart. The cough is more frequent and less hacking, and expectoration is more abundant and mucoid. Paroxysmal attacks of dyspnœa of the true asthmatic type may be present in such cases.

(2) *Emphysema* and bronchitis existing before the attack will increase the gravity of the prognosis, owing to the effect they produce upon the right ventricle, and to the tendency to cardiac failure and pulmonary engorgement thereby entailed.

(3) *Pleuritic effusion*, followed as it often is, especially in children, by *empyema*, is a condition likely to be overlooked unless careful daily examination of the chest is made. The effusion may be primarily sero-fibrinous or hæmorrhagic. The effect of this complication upon the course of the disease is to render the crisis incomplete, and to delay convalescence.

When tubular breathing is followed by complete absence of respiratory sounds, vocal fremitus and resonance being diminished or absent, it must not hastily be concluded that an effusion has taken place into the pleura. In a case of apex pneumonia recently observed the occurrence of these signs led to puncture with a trocar. No fluid was withdrawn, and post-mortem the lung was found to be solid, the bronchi throughout the area of consolidation being filled with solid fibrinous cylinders—'massive pneumonia.' Such cases are usually of a severe type, and often have a fatal termination. Similar physical signs have, however, been observed, when it was proved on post-mortem examination that the bronchi did not contain solid casts. The obstruction in such cases may have been only temporary, and due to the presence of mucus. The position of the cardiac apex is an important guide to the presence of fluid. In children tubular breathing often continues audible, notwithstanding the presence of a considerable effusion in the pleura.

(4) *Pericarditis* is a grave complication of pneumonia, more than half the cases in which it occurs proving fatal (Huss). Like that last described, it is apt to be overlooked, but its presence may be suspected from the position assumed by the patient, who often prefers to be propped up by pillows instead of lying flat upon his back as in uncomplicated cases. A 'to-and-fro' friction sound will be present, and remain until the effusion has become extreme. It must be remembered that an inflammation of the pleura overlying the heart may give rise to a friction sound having a 'to-and-fro' character.

In cases complicated by pericarditis the crisis is often incomplete and the pyrexial period prolonged. The relative frequency of the association of pericarditis with right and left-sided pneumonia is considered under Complications (*vide* p. 209).

(5) *Myocardial* changes are present in all cases of severe pericarditis, but they may occur independently. Their presence may be suspected when there is marked weakness of the heart's action, with evidence of engorgement and œdema of the lung.

(6) Acute *endocarditis* is a somewhat rare but serious complication of pneumonia; it may be either of the simple or ulcerative variety (see p. 209), and either primary or engrafted upon chronic endocardial changes.

(7) *Delirium tremens* is one of the most grave complications of pneumonia, as it implies excessive indulgence in alcohol, with the defective resisting power and visceral changes which such a habit necessarily induces.

(8) *Meningitis* is a rare but almost invariably fatal complication.

(9) *Chronic nephritis* tends markedly to increase prostration, and to mask the more acute symptoms of pneumonia. Granular kidney, as already stated, is frequently found after death from pneumonia, when the presence during life of chronic renal disease has been unrecognised.

(10) *Parotitis* deserves mention on account of its gravity, recovery in cases being exceptional when this complication is present.

A painful and rapidly extending swelling appears behind the ramus of the jaw; suppuration quickly occurs, and may be followed by gangrene. The affection may appear during the later part of the pyrexial period, or after the crisis. It is most commonly met with in patients over sixty years of age.

It may be mentioned here that parotitis of this type differs from a similar affection occurring as a complication of cases of abdominal disease, as in such cases suppuration is rare; and if suppuration does not occur, the prognosis is favourable.

Acute nephritis, jaundice, laryngitis, tonsillitis, and other complications are scarcely of sufficient importance to require description.

J. K. F.

CHAPTER XVII

PNEUMONIA—*continued*

DIAGNOSIS. PROGNOSIS. TREATMENT

PAGE
Diagnosis 226
Prognosis 229
Treatment 230

DIAGNOSIS

In typical cases the diagnosis of pneumonia rarely presents much difficulty, but if the patient is seen at a very early period it may be necessary to wait until the physical signs appear before expressing a positive opinion. A sudden and rapid rise of temperature, persisting after a severe rigor, should suggest pneumonia, if there is no affection of the throat to account for the symptoms. The appearance, decubitus, cough, sputa, altered pulse-respiration ratio, and severe pain in the side, together with the physical signs of consolidation of the lung, constitute a clinical picture which cannot well be mistaken.

Pneumonia of the apex is much more likely to be overlooked than basic pneumonia. This arises partly from the fact that it is comparatively rare, and therefore, although the symptoms suggest the correct diagnosis, attention is directed to the base instead of the apex; partly from the trivial fact that, when a patient sits up in bed and his night-gown is pulled up to his shoulders, the small area of the upper lobe (see fig. 98, p. 352) which abuts posteriorly is covered and may escape examination. We have known apex pneumonia to be recognisable by physical signs in the supraspinous fossa for two days before any certain signs were present beneath the clavicle.

The physical diagnosis of **consolidated lung** from **fluid in the pleural cavity** may be very simple, or present difficulties only to be solved by puncturing the chest; but these difficulties do not often arise in the diagnosis of *pneumonia* from *acute pleurisy with effusion*. This is not a distinction without a difference, for consolidation of the lung may be due to other causes than pneumonia, and fluid in the pleura is not necessarily the result of acute pleurisy.

Before discussing the question, we desire to emphasise two facts: first, that *inspection is one of the most important steps in physical diagnosis*; secondly, that *the position of the cardiac apex* is the key to the diagnosis of nearly all the affections both of the lungs and of the heart.

We shall compare two typical cases—one of pneumonia of the right lower lobe, and another of acute pleurisy with effusion on the right side.

In pneumonia, on *inspection*, the movement of the affected side is limited, but less so than in pleurisy with effusion, whilst the increased movement of the opposite side in the latter condition is more marked than in pneumonia. There is no rounding of the side, or flattening or bulging of the intercostal spaces in pneumonia, and the apex beat of the heart is not displaced.

Palpation.—Vocal fremitus is usually increased over consolidated lung, but diminished over the site of a pleural effusion. If, however, pleural adhesions over limited areas existed prior to the attack of pleurisy, the lung may be held in partial contact with the chest wall, and may act as a conductor of the laryngeal sounds to any point, or yield normal or modified breath sounds.

The liver is not displaced downwards in pneumonia, it may be in pleurisy with effusion, but only if there is a positive pressure within the cavity of the pleura.

Percussion.—The percussion note is dull in both affections, but the loss of resonance is more complete in pleurisy, and the sense of resistance is greater. In pneumonia the area of dulness corresponds to that of the lower lobe, whereas in pleurisy with effusion the upper limit of the dull area presents a curve with the highest point in the axilla. Sub-tympanitic resonance (skodaic note) may be elicited beneath the clavicle in both affections, but is more often present in pleurisy with effusion. It is, however, absent if the effusion is so large in amount as to completely fill the pleural cavity.

Auscultation.—The breath sound is tubular in pneumonia or may possess a metallic quality; whereas in pleuritic effusion the breath sound is absent as a rule, but tubular breathing may be audible through an effusion, a condition more often present in children than adults. Crepitation may be present in pneumonia but not in pleurisy, and is well marked. A pleural friction sound may be present in pneumonia, accompanied, as it usually is, by pleurisy, but will not be audible over the site of a pleural effusion, although it may be present above the upper level of the fluid. Bronchophony is usually well marked in pneumonia; in pleurisy, whispering pectoriloquy or œgophony may be present.

Mensuration.—There is little or no increase in the measurement of the affected as compared with the unaffected side in pneumonia, but there may be a marked increase in pleurisy with effusion.

The above is not intended as an exhaustive statement of the physical signs which may be met with in the two affections, but only of such as are present in typical cases.

Lobar caseous tuberculosis—by which term (*vide* p. 336) we indicate a rare affection in which a whole lobe of a lung becomes rapidly solid from tubercular disease—may simulate pneumonia both in symptoms and signs, and it often happens that the nature of the case is not at first recognised. The absence of 'crisis' and the continuance of pyrexia and consolidation suggest at a later period an examination of the sputum for tubercle bacilli, the discovery of which at once settles the diagnosis. In doubtful cases repeated examinations should be made, as until softening commences the bacilli will probably not be found.

A large **hæmorrhagic infarction** of the lungs, with acute pleurisy and effusion, occurring in a case of chronic valvular disease of the heart, is a condition which we have on several occasions seen mistaken for pneumonia.

The first point which should attract notice in such a case is that the patient will almost certainly be sitting up in bed. It is a good rule never to come to a diagnosis of *uncomplicated* pneumonia when the patient is in that position. The 'erect posture,' of a well-known and most valuable textbook of medicine, as descriptive of the typical decubitus in pneumonia is surely an oversight.

Dulness on percussion over the affected side and marked tubular breathing over the consolidated area are usually present. The vocal fremitus may be absent towards the spine. Such cases are often too ill to bear the movement which is required to carry out a careful examination of the back of the chest. The cardiac apex may be displaced, but it should be possible to determine the presence of the valve lesion if that is not already known.

Typhus fever is so seldom seen nowadays that it is rare for it to be mistaken for pneumonia.

Typhoid fever.—The asthenic forms of pneumonia, which are due in many cases to septic infection and are accompanied by marked prostration, are liable to be mistaken for typhoid fever. The presence of herpes on the lips, and of signs of consolidation of the lung, are the most important distinguishing features of pneumonia as compared with typhoid fever. Pneumonia may, it is true, occur as a complication of typhoid, but it does so in the later stages of that disease, whilst the question of diagnosis almost always arises at an early period of the case.

Meningitis.—The cerebral symptoms, such as headache and delirium, by which the onset of pneumonia, especially of the apex of the lung, is sometimes accompanied in children may for a time suggest a diagnosis of meningitis, either simple or tubercular. It is important to bear in mind that in most acute diseases headache as a rule subsides when delirium appears, whereas in meningitis headache and delirium are present together. The other symptoms which mark the stage of 'cerebral irritation' in meningitis, such as the peculiar cry, the curled-up attitude, the retraction of the head and of the abdomen, the vomiting without cause, and the intolerance of light, are usually absent in pneumonia.

From **œdema** of the lung pneumonia can usually be distinguished by the absence of fever and of the signs of complete consolidation. Although the resonance of the percussion note may be impaired at one or both bases, there is not the same degree of dulness as in pneumonia. The breath sounds in œdema are feeble. Crepitation indistinguishable from that of pneumonia may be present.

From **collapse of the lung**, a condition which may in children affect a whole lobe, the diagnosis is often difficult. The temperature is lower than in pneumonia. The affected side in cases of collapse is retracted, the breath sounds are absent; or if, as is sometimes the case, they are present and of bronchial or tubular quality, the sound gives the impression of being conducted from a distance. The vocal fremitus and resonance are diminished and adventitious sounds are absent.

PROGNOSIS

In forming a forecast of the probable issue of a case of pneumonia many factors have to be considered, and much depends upon the capacity to recognise the signs of danger, which can only be acquired by careful observation at the bedside.

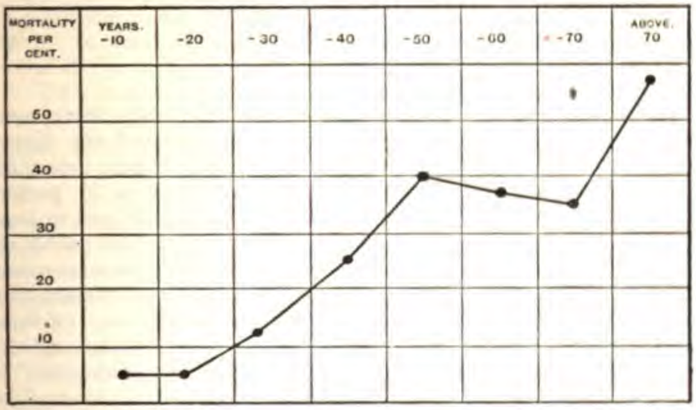


FIG. 74.—CURVE OF MORTALITY FROM PNEUMONIA AT DIFFERENT AGE PERIODS (BASED ON ANALYSIS OF 2,038 CASES)

(From 'Pneumonia,' by Sturges and Coupland, p. 141)

'It is probable that the smallest mortality from lobar pneumonia period of life whatever occurs in early childhood.'[1]
fact that the disease is relatively more fatal in females than

---

[1] *Pneumonia*, Sturges and Coupland, 2nd ed. p. 135.

inducing natural sleep renders the use of hypnotics unnecessary; that the improvement in the pulmonary condition obviates the necessity for venesection; and, lastly, that it secures a more rapid convalescence.

We approached the trial of this method of treatment with a strong bias in its favour, the result of long experience of the extreme value of antipyretic measures in typhoid fever, the clinical aspect of that disease having, in our opinion, been almost revolutionised by their use. Notwithstanding this, however, we are now unable to give it more than a qualified support in pneumonia. Dr. Lees has strongly advocated the use of the icebag, and we have epitomised above the claims which he puts forward in its favour. That the application of an icebag lowers the temperature, and in some cases gives marked relief to pain, may be readily admitted, and we have frequently observed that sponging the surface of the body for from ten to fifteen minutes at a time with water at a temperature of 80° F. is grateful to the patient, and that it lowers the temperature, relieves restlessness, and promotes sleep.

It may, we think, be fairly claimed for the moderate and judicious use of antipyretic measures in pneumonia that the results obtained are as good as those from any other mode of treatment at present adopted; but we are still unconvinced that the course of the disease is decidedly influenced by the systematic application of cold to the surface of the body. The more active antipyretic drugs, in doses sufficient to influence the temperature, are decidedly depressant in their effects, and should not in our opinion be employed. Possibly in an early stage of the disease they may sometimes give relief by inducing sweating, and in small doses in combination with quinine they may be useful as an adjunct to sponging.

For the treatment of *hyperpyrexia*—by which is meant a temperature of 105·8° and over—the ice-cradle is preferable to the cold bath, as its use does not disturb the patient. If, however, the temperature cannot be controlled by this means, the ice-pack should be used, or a bath at 80° F. given for about ten to fifteen minutes. If the course of the fever is carefully watched, and sponging resorted to when the temperature rises above 104° F., the necessity for more severe anti-pyretic treatment may be obviated.

The tendency to *cardiac failure*, which is evidenced by a feeble and irregular pulse and signs of increasing distension of the right ventricle with blueness of the lips, may be met by the early use of oxygen, as recommended by Sir R. Douglas Powell, who has pointed out[1] that this most valuable agent is generally employed too late in the disease to be of any but temporary service. Carbonate of ammonia and digitalis should be given at the same time with a like object. In a very severe case of double pneumonia recently under our care in a boy aged 14, oxygen was used at an early period, and its beneficial effect upon the pulse and respiration was most marked.

---

[1] *Brit. Med. Jour.* Nov. 9, 1895, p. 1154.

It is under such circumstances that *stimulants* are needed, and they may be required in large doses, *e.g.* six, eight, twelve, or even twenty ounces of brandy in the twenty-four hours. Whilst fully convinced of the necessity and value of stimulants in the treatment of pneumonia, increasing experience has convinced the writer that the practice, in which he was educated, of administering them in large doses and as a matter of routine in all cases of pneumonia, is both unnecessary and harmful, and that much injury may be done by the over-stimulation of a weak heart. By giving alcohol and digitalis together, the temporary stimulant effect upon the heart of the former and more permanent and tonic effect of the latter are best obtained.

In addition to the measures already described, strychnine by hypodermic injection (gr. $\frac{1}{50}$–$\frac{1}{30}$) and caffeine are valuable cardiac tonics which may be used at the same time as inhalations of oxygen in endeavouring to maintain a failing heart.

Violent delirium at a late stage of the disease is, as already stated, a most dangerous condition, and is usually an indication for the administration of stimulants. Sir R. Douglas Powell has laid stress upon the value, under these circumstances, of a large dose or frequently repeated doses of alcohol given with food, followed by a hypodermic injection of morphine and atropine. In a severe case of apex pneumonia recently under our care, with marked but not violent delirium and almost incessant hiccough, a hypodermic injection of morphine was followed by sleep, the gradual disappearance of the hiccough, and by recovery. Its administration appeared to be the determining factor in the case.

Pericarditis complicating pneumonia is always an indication that the attack is of a severe type, and calls for the administration of stimulants.

Diarrhœa is rarely a prominent symptom; if severe, it may be checked by the administration of Dover's powder.

When at a late stage of the disease there is evidence of *extending implication of the lung* with prostration, perchloride of iron in large doses has been found of service.

Cyanosis and signs of over-distension of the right side of the heart, with epigastric pulsation and prominence of the jugular veins and a small and irregular pulse, are indications for *venesection*, and relief is generally given when six or eight ounces of blood have been withdrawn. The improvement is perhaps most obvious in cases accompanied by or following upon bronchitis, but unfortunately it is as a rule of only short duration.

Dr. G. W. Balfour strongly advocates the use of chloral (gr. 10) combined with infusion of digitalis (℥ss) every four hours during the period of pyrexia, double the quantity of chloral being given for the first dose. It is claimed for this treatment that it gives the patient most relief from his sufferings, favours an early crisis, and has no drawbacks; and that if, by its use, the mortality is not diminished, it is certainly not increased. By the combination with gitalis, the undoubtedly depressant effect of chloral upon the heart and respiration is counteracted, but when not so combined its administration is not unattended with danger. We are unable to speak from personal experience of the value of this mode of treatment.

After the crisis, if, as is often the case, resolution makes slow progress, or if the lung continues solid, or a pleural effusion should remain, equal parts of the liniment and tincture of iodine should be painted over the side affected. This may be done every morning for several successive days until some irritation is produced, and subsequently repeated at longer intervals according to the condition of the skin, the susceptibility of which to the action of iodine varies greatly in different individuals. If resolution is long delayed, the syrup of the iodide of iron with quinine may be given. A cotton-wool jacket should be worn until recovery is complete.

With convalescence, appetite quickly returns, and should be satisfied by a liberal diet. Tonics such as iron and quinine should then be prescribed. After an attack of pneumonia, a stay for a time in a bracing air is generally advisable.

The treatment of abscess of the lung and of pulmonary gangrene is considered elsewhere (*vide* pp. 243 and 250).

Antitoxic treatment.—The increasing evidence in favour of its microbic origin has naturally led to the inclusion of pneumonia amongst the diseases to which serum treatment is applicable, and every one must share in the hope that, at no distant date, this mode of dealing with the disease may come within the range of practical therapeutics.

There are two possible sources of the serum. It may be obtained from the blood of a patient who has recently passed through the crisis of the disease or from an animal rendered immune to the disease by inoculation with a gradually increasing dose of the pneumococcus. It is naturally to the latter source that we must look for a supply of the material if the treatment becomes available for general use. At present the method can hardly be said to have passed beyond the experimental stage. Dr. Washbourn has succeeded in 'standardising' the antitoxin, *i.e.* in obtaining a serum of uniform strength, so that the dose administered is accurately known. This is an important step in advance, and a certain number of severe cases have been thus treated, and recovery has followed. Whether this favourable result is to be attributed to the treatment adopted it is as yet impossible to say, but the results appear to be decidedly encouraging. No ill effects have followed its use.

Recent experience has shown how extremely undesirable it is that a remedy of this nature should be generally employed until it has been most carefully tested by persons competent to form an opinion of its value. It is to be hoped that the mistake will not be repeated in the treatment of pneumonia.

Local applications.—The severe pain which often accompanies the onset of pneumonia is probably due to the stretching of the inflamed pleura by the engorged lung. It may be intense, when no friction sound is audible. As already stated, it may be relieved by the application of an icebag, but we confess to a preference for the use of leeches followed by the application of a large linseed-meal poultice. Poultices are, it is true, somewhat out of fashion, but we have seen no reason to discard them, except in some cases in children, when their weight is an objection, as tending to impede the movements of the chest. Patients frequently express their appreciation of them, and we cannot remember to have heard a complaint against a well-made poultice. They should be large and light, properly secured in position, and changed every two or three hours during the day, and at rather longer intervals during the night. These details are important, and, if attention to them cannot be secured, it is far better to cover the affected side with a layer of cotton wool heated at the fire and sprinkled with a stimulating liniment. When for any reason the use of poultices is discontinued, a cotton-wool jacket should be worn.

J. K. F.

CHAPTER XVIII

CHRONIC AND SUBACUTE PNEUMONIA

(LOBAR TYPE)

PAGE
Differentiation from acute pneumonia 237
Morbid anatomy 237
Etiology 239
Duration 239
Prognosis 239
Diagnosis 239
Treatment 240

It has long been, and in the opinion of some writers is still, an open question whether an attack of pneumonia is ever followed by permanent induration of the lung. That a point of this nature should remain undecided in connection with a disease of such frequent occurrence as pneumonia is in itself a proof that such a sequence of events must be infinitely rare. We entertain no doubt that it does occur, but it can hardly fall to the lot of any individual to observe more than a very few cases of the kind. Mention has already been made (*vide* p. 230) of a case which was observed by the writer in a youth who had had seven previous attacks of pneumonia, for most of which he had been an in-patient in the Middlesex Hospital, and who left convalescent after the last attack, but with signs of induration of the apex of the left lung. The breath sounds at this apex were almost cavernous in quality, although there had been no signs of softening. They were probably due to the dilatation of a bronchus.

In the few undoubted cases of this kind recorded in medical literature, bronchiectasis has generally been found at the autopsy, death having in some been due to an acute septic broncho-pneumonia followed by gangrene.

In others, bronchiectasis has been absent, or present to a very slight degree only, and the condition has resembled that found in the cases about to be described, the clinical history of which differs somewhat from that of true pneumonia.

SUBACUTE INDURATIVE PNEUMONIA (Primary Parenchymatous Pneumonia)

The former of the above terms, which appears to be preferable, was suggested by Dr. Percy Kidd,[1] the latter by Buhl, and is adopted by Heitler.[2]

These cases present certain features hitherto rarely recognised in addition to their mode of termination, by which they are differentiated from acute pneumonia.

The chief points of difference may be thus summarised :—

(1) The onset, although sudden, and accompanied by chills or rigors, is not marked by the single severe rigor of pneumonia.
(2) The illness immediately following is not of the severe and acute character of pneumonia. The patient may, for example, be able, although feeling ill, to leave his bed on the second day. The prominent symptoms are fever, dyspnœa, a rapid pulse, and cough. Herpes may be present.
(3) The accompanying pyrexia is not so marked as in pneumonia, and there may be complete remission of the fever for short periods.
(4) There is no critical fall of temperature.
(5) The sputum may contain blood, but rarely presents the true rusty character. It is abundant in quantity, and has a decided tendency at a later stage to become fœtid.
(6) Diarrhœa is a more common symptom than in the acute disease.
(7) The physical signs of consolidation of the lung do not appear to be so rapidly developed as is the case in pneumonia.
(8) In pneumonia the constitutional symptoms tend gradually to diminish in severity when the exudation is absorbed, even though the lung has remained solid for a prolonged period; but in subacute indurative pneumonia consolidation persists, while the general condition of the patient becomes progressively more unfavourable, and putrid bronchitis and gangrene are very liable to precede a fatal termination.

Morbid anatomy.—Pleural adhesions are present over the consolidated lung. They may be dense and fibrous, and the chest wall may be retracted over the corresponding area. Fibrosis and xcavation are the chief lesions present. The fibroid change in e lung is lobar in distribution, but may be present in more than gle lobe and in various stages, and both lungs may be affected. onsolidated part is airless, shrunken, and of firm consistence, granular on section, and either red, grey, slate-grey, or almost white in colour.

---

[1] *Lancet*, April 5, 1890.

[2] *Wiener Med. Wochenschr.* 1884, 1886.

These changes in colour represent stages of the morbid process.

The consolidation may not be uniform throughout a lobe, patches of air-containing lung intervening between areas of induration. When cavities are present, the appearances they present vary with the duration of the lesion. If it is of somewhat recent date, softening may be in progress at various points, and the cavity walls are ragged and possibly gangrenous, a result of the necrosis of the indurated tissue. More chronic cavities present a smooth interior, and are limited by a fibrous capsule. Broncho-pneumonia and putrid bronchitis may result from the inhalation of the decomposed contents of cavities. These changes may occur independently of bronchiectasis.

On *microscopical examination* the changes observed are mainly of two kinds: the one showing that an alveolar exudation is in process of organisation, the other that the alveolar walls and the interstitial tissue of the lung are undergoing fibrous thickening.

The early stage of the former process is best seen in the areas of red induration, and is thus described in Dr. Kidd's paper:

'The alveoli contained roundish masses of a coarsely granular opaque material, in which a few nuclei were visible. In places these plugs at their centre showed a delicate meshwork of fibrinous threads, and in rare instances consisted mainly of fibrinous matter. In every case the alveolar plugs were ensheathed in a more or less complete investment of young connective tissue, separating them from the alveolar walls.'

Scanty capillary blood-vessels could be traced in the intra-alveolar connective-tissue capsules, and in some instances they were clearly connected with the capillary network in the walls of the vesicles. The infundibula were filled with recent fibrinous exudation, containing a few leucocytes and red blood-corpuscles, without any connective tissue investment.

In the grey consolidation a more advanced stage of the same condition was found. The air sacs were stuffed with rounded masses of connective tissue, consisting of a central core of round and spindle cells with a meshwork of delicate fibres surrounded by a more distinctly fibrous outer zone.

The interstitial tissue showed a slight diffuse thickening, more marked in the grey than the red area; but this change was insignificant as compared with the intra-alveolar growth. The thickening of the alveolar walls was due to the presence of a moderate degree of small-celled infiltration.

The interstitial changes are due to fibrous thickening of the alveolar walls, and of the interlobular septa and peribronchial and perivascular connective tissue.

The appearances are illustrated in the following drawing of a section taken from the case described by Dr. Percy Kidd, and kindly lent by him.

The *associated lesions* likely to be met with are hypertrophy and dilatation of the right side of the heart, chronic nephritis, fatty or nutmeg liver, and amyloid changes in various viscera.

Etiology.—No definite statements can be made as to the etiology of this affection. Alcoholism has been noted in some cases.

The **duration** of the cases of this nature hitherto recognised has varied from five weeks to nine months, but it is possible that the condition may be still more chronic, and may then assume the characters described under Pulmonary Fibrosis (*vide* p. 260). Death has been due to cardiac failure, diffuse septic broncho-pneumonia and gangrene, or chronic renal disease.

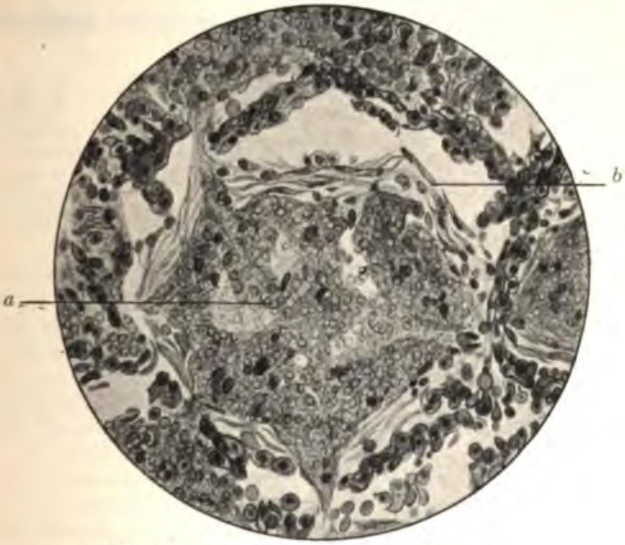


FIG. 75.—SUBACUTE INDURATIVE PNEUMONIA

a, granular matter and capillary vessels in alveolus, with nucleated cells ; *b*, connective tissue ensheathing exudation

Prognosis.—This is necessarily very grave. When the sputa have become fœtid a fatal termination may be looked for within a short period.

Diagnosis.—Probably the first step in the recognition of such a case will be failure to find tubercle bacilli in the sputum. The diagnosis of bronchiectasis will probably next su[illegible]t itself, and will be excluded with greater difficulty, but
attention to the history of the case should suffice for this.
ace may be made to the article on that subject, and par-
ly to the stages through which, in many cases, that affection
(*vide* p. 134). There will be no history of such stages, but,
contrary, there will be one of the more or less sudden onset
ase of a somewhat severe character. The lobar distribution
sions may suggest that it is a case of pneumonia, but

if diffuse septic broncho-pneumonia or gangrene is present, little help will be obtained from the observation of the area affected.

Treatment.—The general treatment should be tonic and supporting, and the patient should be careful to avoid exposure to conditions likely to give rise to bronchial catarrh. If the sputa and breath are fœtid, the use of the creasote vapour bath, as described in the chapter on Bronchiectasis, would be suitable. If the site of a large single basic cavity produced by necrosis, and possibly gangrene of lung, could be discovered, an attempt should be made to drain it as described in the chapter on the Surgical Treatment of Pulmonary Cavities.

Any complications which arise should be treated upon general principles.

J. K. F.

CHAPTER XIX

ABSCESS OF THE LUNG

	PAGE
Etiology	241
Symptoms	242
Physical signs and diagnosis	242
Course	242
Prognosis	243
Treatment	243

A MORE or less circumscribed collection of pus within the lung may occur as a secondary result of a great variety of affections. In the majority of conditions to which the term is applied, the similarity of the lesion to an ordinary 'abscess' is, from a pathological point of view, not very close.

Etiology.—An abscess may be met with in connection with—

(1) Pneumonia followed by necrosis and softening of a considerable area of the consolidated lung.
(2) Pulmonary tuberculosis, when a large caseous area undergoes rapid disintegration.
(3) Chronic non-tubercular cavities.
(4) Bronchiectasis, either with or without ulcerative changes in the bronchial walls and surrounding tissues.
(5) Suppuration of an hydatid cyst.
(6) Softening of a pyæmic infarction.
(7) Pulmonary embolism followed by softening.
(8) Perforation of the lung by a malignant growth in the œsophagus.
(9) Suppuration of bronchial glands extending into the lung.
(10) Empyema rupturing into the lung.
(11) Perforation of the lung by a mediastinal abscess.
(12) Perforation by an abscess extending through the diaphragm.
(13) Injuries of the lung.

The precise nature of the lesion present in most of the above conditions is fully described elsewhere in this work; it will therefore only be necessary to consider here those to which the term 'abscess' of the lung is commonly applied.

Abscess of the lung may be either single or multiple. The former variety is met with in pneumonia, and as a result of injuries, such as fractures of the ribs, or wounds penetrating the lung, and in some other conditions. Multiple abscesses are generally pyæmic in origin, and are due to the softening of infarctions or to septic broncho-pneumonia.

Abscesses of this nature are usually of small size, and, beyond the occasional presence of limited patches of pleural friction and possibly a small amount of fœtid expectoration, give rise to no symptoms which can be distinguished from those of the general condition, and in some cases even these symptoms are absent.

Symptoms.—*Pyrexia.*—During the period of formation of an acute pulmonary abscess, as, for example, in pneumonia, there is continued fever, the temperature [illegible] at a high level; subsequently the pyrexia is of the hec[illegible]monly observed when matter is retained within the b[illegible]g this period rigors may occur. The discharge of [illegible] generally followed either by a fall of temperature [illegible] disappearance of the pyrexia.

Expectoration.—The sudden [illegible]on of a large quantity of pus, in which, either with the na[illegible] eye or by the aid of the microscope, lung tissue is found, is the most characteristic symptom. Micro-organisms varying in nature with the condition, but generally either pneumococci or streptococci, are invariably present. The expectoration is often, but not always, fœtid; but, if free from odour when first discharged, it is apt to become fœtid at a later period if the drainage of the cavity is not free.

Physical signs and diagnosis.—The former are extremely variable. When signs indicating the presence of a cavity are discovered shortly after the evacuation of a large quantity of pus, there cannot be much doubt as to the nature of the case; but, previously to this, dulness on percussion and absence of breath sounds may have suggested a diagnosis of pleural effusion. The absence of displacement of the cardiac apex and increased instead of diminished vocal fremitus should, however, prevent this error. Softening in several areas is accompanied by large crackling râles. When, instead of rupturing into a bronchus, the abscess perforates the pleura or the pericardium, it will be very difficult to determine the exact condition present. If situated near the surface of the lung, the pleura will certainly become involved, and the presence of a pleural friction sound may suggest the nature of the case when the import of other signs is doubtful.

An abscess has been known to perforate the intercostal muscles, and to give rise to subcutaneous emphysema, and in rare cases to discharge itself externally. Very rapid softening of an area of caseous tuberculosis may simulate abscess of the lung, but the true nature of the condition is revealed by the discovery of tubercle bacilli in the expectoration.

The **course** of a case of pulmonary abscess will depend greatly the nature of the primary condition on which it depends.

If it is a result of pneumonia the discharge of the contents may be followed by a gradual disappearance of symptoms and signs, and by the cicatrisation of the cavity, but such a favourable issue is extremely rare. More often death occurs from gradual exhaustion, or septic broncho-pneumonia caused by inhalation of the secretion from the abscess cavity.

The **prognosis** is in all cases very grave, but, as already stated, the condition is not necessarily fatal.

Treatment.—The indications are to increase by all possible means the resisting power of the patient and to bring about local conditions which may, when such a result is possible, lead to the contraction and ultimate obliteration of the cavity. The former is fulfilled by the administration of quinine in large doses and of perchloride of iron, with plenty of nourishing food, and by placing the patient under the most favourable hygienic conditions. The first step towards the latter is to secure free drainage from the cavity. When there is clear evidence of the presence of an abscess cavity within the lung, the question of surgical interference will arise, and will be decided by a careful consideration of the general condition of the patient, the nature of the primary disease, the site of the cavity, whether apical or basic, single or multiple, the possibility of securing free drainage, and the condition of the overlying pleura. These points are fully considered in the chapter on the Surgical Treatment of Pulmonary Cavities (*vide* p. 416 ff.). Abscesses due to pyæmia are, however, practically beyond the reach of treatment.

If, as is generally the case, the sputa are fœtid, creasote may be given internally and by inhalation, either on an oro-nasal inhaler or, if the patient can bear it, by means of the creasote vapour bath. Cough which is effective in expelling secretion should not be checked by sedatives, as both secretion and cough will diminish with improvement in the local condition.

former variety is met with in pneumonia, and as a result of injurie such as fractures of the ribs, or wounds penetrating the lung, a in some other conditions. Multiple abscesses are generally pyæm in origin, and are due to the softening of infarctions or to sep broncho-pneumonia.

Abscesses of this nature are usually of small size, and, bey the occasional presence of limited patches of pleural friction possibly a small amount of fœtid expectoration, give rise to no s ptoms which can be distinguished from those of the general condit and in some cases even these symptoms are absent.

Symptoms.—*Pyrexia.*—During the period of formation acute pulmonary abscess, as, for example, in pneumonia, the continued fever, the temperature ranging at a high level; s quently the pyrexia is of the hectic type commonly observed matter is retained within the body, and during this period may occur. The discharge of the pus is generally fol either by a fall of temperature or by the disappearance pyrexia.

Expectoration.—The sudden expectoration of a large tity of pus, in which, either with the naked eye or by the ai microscope, lung tissue is found, is the most characterist ptom. Micro-organisms varying in nature with the co but generally either pneumococci or streptococci, are in present. The expectoration is often, but not always, fœtid free from odour when first discharged, it is apt to beco at a later period if the drainage of the cavity is not free.

Physical signs and diagnosis.—The former tremely variable. When signs indicating the presence of are discovered shortly after the evacuation of a large q pus, there cannot be much doubt as to the nature of the previously to this, dulness on percussion and absence sounds may have suggested a diagnosis of pleural effu absence of displacement of the cardiac apex and increas of diminished vocal fremitus should, however, prevent Softening in several areas is accompanied by large crac When, instead of rupturing into a bronchus, the absce the pleura or the pericardium, it will be very difficult the exact condition present. If situated near the su lung, the pleura will certainly become involved, and th a pleural friction sound may suggest the nature of th the import of other signs is doubtful.

An abscess has been known to perforate the interc and to give rise to subcutaneous emphysema, and in discharge itself externally. Very rapid softening caseous tuberculosis may simulate abscess of the lu nature of the condition is revealed by the discovery [illegible] tion.

is evidence of an attempt on the part of the organism to limit the process, resembling that which, in the case of gangrene of a limb, leads to the formation of a 'line of demarcation.'

Within the limiting zone the lung tissue may persist as a black, grey, or greenish mass, or it may have broken down and have been in part expectorated. If a cavity has resulted the walls are ragged and shreddy in recent cases, but, if the duration of the process has been more prolonged, the necrosed tissue may have separated as a slough, and the walls may consist of a smooth layer of granulation tissue. In cases of a still more chronic nature there may be a well-defined zone of induration. The contents of the cavity are horribly fœtid, grey, black, blackish-green or chocolate in colour, and either pulpy or diffluent in consistence. If recent hæmorrhage has occurred the cavity may be filled with blood.

The bronchi and large vessels of the affected area may have been destroyed in the process, or the vessels, filled with thrombi, may be found amongst shreds of necrosed tissue still adherent to the ragged walls. The neighbouring bronchi are acutely inflamed and usually contain fœtid material similar to that found within the cavity.

If the lesion is situated near the surface, the pleura over the affected part will be acutely inflamed and covered with lymph. The gangrenous area may be very small or it may, as in some cases following pneumonia, involve a considerable part of a lobe; when of embolic origin, it is often wedge-shaped.

Diffuse gangrene.—This, which is the rarer form of the lesion, is characterised by the absence of a limiting zone of inflammation, the gangrenous area merging with the surrounding lung tissue.

The affected part is of a grey, greenish, or black colour; generally, but not invariably, of a fœtid odour, and may be of any consistence from that of hepatised lung to that of a pulp, or it may present the appearance of blackish grumous fluid occupying a ragged cavity. The surrounding lung is œdematous, and generally emits a fœtid odour. If the lesion is situated near the surface, the pleura will probably have undergone extensive necrosis and gangrene. Empyema and pyopneumothorax may be present.

The bronchi and vessels of the affected area may still be recognisable, and the latter often contain thrombi.

Micro-organisms of various kinds have been found in gangrenous areas in the lungs. The staphylococcus pyogenes aureus is believed by Bonome[1] to be the primary cause of the necrosis of the pulmonary tissue, as injections of pure cultivations of the staphylococcus pyogenes aureus into susceptible animals are said by the same observer to be followed by gangrene of the lungs. Babès[2] has described a mobile rod-shaped organism from ·8 to 1·5 μ in length, which was present in the sputa of a case of pulmonary gangrene, and which, when injected into susceptible animals, gave rise to septicæmia. Cultivations of the organism were free from odour. Leptothrix, aspergilli, sarcinæ, mucor mucedo, and other organisms have also been found. It is, however, improbable that pulmonary gangrene is due to the presence of any special organism; it would rather appear to be dependent upon the combined action of several saprophytic organisms.

---

[1] *Deut. Med. Woch.* 1888, No. 52.

[2] Cornil et Babès, *Les Bactéries*, 1890, tome i.

Etiology.—The primary conditions leading to pulmonary gangrene may be classified under the following headings, viz.: (*a*) lesions of the bronchi; (*b*) lesions of the lungs and mediastinum; (*c*) lesions of the vessels; (*d*) extension from lesion of neighbouring viscera; (*e*) wounds of the lungs or thorax; (*f*) general conditions.

(*a*) LESIONS OF THE BRONCHI.—The retention of putrid secretions within the bronchi is not infrequently the cause of gangrene of the lungs. In the majority of cases originating in this way the bronchi are dilated; but, as was stated in the description of *putrid bronchitis*, dilatation is not an essential condition, although it is rarely absent when gangrene supervenes. In bronchiectasis the expectoration may be fœtid for years without the occurrence of gangrene; but if, as not uncommonly happens towards the end of the case, a diffuse septic broncho-pneumonia should be set up, the lung is very likely to pass into gangrene. Any case of bronchiectasis may terminate in gangrene, which is not necessarily limited to the area in which the bronchi are dilated; decomposing secretion driven by cough into the trachea may be inhaled into the opposite lung and induce gangrene, usually of the lower lobe.

In conditions of paralysis portions of food carrying with them septic organisms may pass unnoticed into the trachea. Diminished sensibility of the larynx, permitting the passage of food and secretions into the trachea, is probably one of the chief causes of the frequent occurrence of pulmonary gangrene in the insane. In melancholia, accompanied by refusal of food, a condition in which pulmonary gangrene has frequently been observed, it may possibly be an indirect result of defective nutrition; but its occurrence has also been attributed to the passage of food into the trachea during forcible eeding. In fœcal vomiting material regurgitated from the stomach may be drawn through the glottis and may induce pulmonary gangrene. Gangrene of the lungs is not a very uncommon mode of termination of cases of epithelioma of the tongue, mouth and œsophagus, and it may occur as a secondary result of gangrene of the mouth and also of diphtheria. Gangrene may follow when a suppurating bronchial gland has established a communication between the œsophagus and the trachea or one of the main bronchi.

(*b*) LESIONS OF THE LUNGS AND MEDIASTINUM.—Gangrene may be induced by any form of inflammation of the lungs if septic organisms develop in the affected area, and, as already stated, broncho-pneumonia is usually present in the various conditions considered in the preceding section.

Pneumonia may undoubtedly pass into gangrene, although many writers have doubted the possibility of the occurrence. The following were the post-mortem appearances in a well-marked case of pneumonia of sudden onset admitted on the third day of the disease and fatal on the eighth day.

'Right lung.—The lower and posterior half of the upper lobe was converted into a sloughy mass, lying in a ragged cavity which was filled with foul greenish opaque fluid. Shreds of gangrenous tissue were adherent to the walls of the cavity. A fairly defined boundary of hepatised lung, mottled, of a greyish red colour, separated the gangrenous area from the surrounding tissue. Some vessels in the walls of the cavity contained fibrinous coagula. The middle lobe was consolidated and in a condition of grey hepatisation. The lower lobe was engorged. The pleura covering the upper lobe was intensely inflamed, studded with hæmorrhages, and covered with patches of lymph.'

It will be observed that the middle lobe of the right lung presented appearances similar to those of the hepatised but non-gangrenous portion of the upper lobe. The hepatisation found in such cases has been stated to be the result and not the antecedent of the gangrene, but it is unlikely that the gangrenous process can induce a true pneumonic consolidation of an adjacent lobe. The factor determining the occurrence of gangrene in cases of pneumonia cannot be exactly defined. A marked lack of resisting power such as that induced by starvation, alcoholism, diabetes, chronic nephritis, or advanced age, appears to be the determining cause in many cases. Assuming the microbic origin of pneumonia it may be held that in some cases there is a double infection and that septic organisms are present in addition to those now believed to be pathogenic of that disease. A similar association of septic and pathogenic organisms in diphtheria has been shown to exercise a marked effect upon the course of the disease.

Broncho-pneumonia in children, especially when secondary to measles, may terminate in gangrene, but such an event is rare. The gangrene usually affects many areas, which have a racemose arrangement.

Chronic pneumonia.—This condition, with which bronchiectasis is very often associated, is liable to be complicated by an acute septic inflammation of the neighbouring lung tissue, and under such circumstances gangrene may supervene.

The mode of production of gangrene in cases of *mediastinal tumour*, aneurism, and growths originating within the lung must be considered doubtful. It has been variously attributed to septic pneumonia secondary to the retention of secretion in the bronchi, and to direct pressure either upon the lung or upon the bronchial vessels or upon the pulmonary plexus. In a case of lympho-sarcoma of the mediastinum recently under our observation obstruction of the main bronchus led to sacculated bronchiectasis throughout almost the whole of the affected lung. This was followed by septic pneumonia and gangrene.

This sequence of events is in our experience not uncommon, and we are disposed to consider the decomposition of retained secretions of far more importance in the production of gangrene than any of the other causes mentioned; it is indeed doubtful whether it may not be considered the only cause.

Pulmonary tuberculosis—This disease very rarely ends in gangrene. Decomposition of secretions within cavities and changes in their walls may give to the breath a fœtid odour, but the special characters observed in gangrene are absent.

(*c*) Lesions of the Vessels.—When an *embolus* derived from a septic source lodges in a branch of the pulmonary artery an infarction results and gangrene may follow; but in the majority of cases a pyæmic abscess forms, the occurrence of gangrene being rare. The area affected usually has the wedge-shaped outline of the ordinary hæmorrhagic infarction.

It must not, however, be thought that the presence post-mortem of coagula in the vessels in gangrene of the lungs necessarily indicates that the process was of embolic origin, as *thrombi* are often formed in such cases. On the other hand, in cases undoubtedly due to embolism the secondary thrombi may render it difficult to recognise the embolus. Common sources of septic emboli in cases of pulmonary gangrene are thrombi in the lateral sinus secondary to caries of the temporal bone, in the uterine veins in cases of puerperal fever, or in veins in the neighbourhood of foci of septic suppuration, such as a sloughing bedsore. Pulmonary gangrene of embolic origin has also been observed, but only rarely, in erysipelas, carbuncle, typhoid fever, and various other morbid conditions.

Fat embolism of the pulmonary capillaries has been shown to be capable of producing gangrene when the emboli are derived from a septic source.

(*d*) Gangrene by extension from neighbouring Viscera.—A septic or gangrenous inflammation situated in the abdomen or thorax may involve the lungs by direct extension and gangrene may follow. Abscess of the liver and subdiaphragmatic abscess are the most common antecedent conditions originating in the abdomen. If in such cases the pleural surfaces are not adherent, empyema may precede the gangrene, which follows perforation of the visceral layer of the pleura. A fœcal abscess may extend upwards to the thorax and be followed by similar results. Cancer and gangrene of the œsophagus may also induce a similar change within the lung.

(*e*) Wounds or Injuries of the Lung or Thorax.—Injuries to the chest wall accompanied by extravasation of blood into the lung tissue may be followed by gangrene. Gunshot wounds of the lung have in some campaigns frequently presented this complication. In some cases of the above nature a true pneumonia appears also to have been present.

(*f*) General Conditions.—Starvation, alcoholism, chronic nephritis, diabetes, and some of the specific fevers, more especially measles and typhoid fever, are the more important general conditions which have been followed by gangrene of the lungs.

Possibly some cases which have occurred after exposure to noxious emanations from sewers and other sources of foul air should be included under this heading.

Symptoms.—Although gangrene of the lungs may occur without revealing itself by the characteristic odour of the breath and sputa, such a condition is, from a practical point of view, of but little importance, as in the absence of such signs its presence is rarely even so much as suspected. Such cases are chiefly met with in the insane, in diabetics, and in the subjects of gangrene from embolism.

Putrefactive decomposition of retained secretions and gangrenous broncho-pneumonia may be present in bronchiectasis without causing fœtor of the breath, if the upper air passages are cut off from communication with the dilated tubes.

Having regard to the very varied conditions under which pulmonary gangrene may occur, uniformity in its clinical manifestations and course is not to be expected. The onset is very often marked by a rigor, which may be repeated; the general condition of the patient is usually one of marked prostration; the involvement of the pleura in the process is evidenced by severe pain in the side. The progress of a destructive change in the lungs is attended by a liability to hæmoptysis, which may be very profuse. Hæmoptysis has indeed in some cases been the first symptom observed.

The changes in the general aspect of patients suffering from bronchiectasis or pneumonia when pulmonary gangrene sets in are dealt with on p. 134 and p. 222.

The peculiar odour of the breath and sputa in cases of gangrene is readily appreciated but difficult to describe. The expectoration resembles that of bronchiectasis in the tendency to settle into three layers, after standing for about twelve hours, but differs from it in the variety of colours which may be present. These are probably owing to the intermixture of blood pigment and the chemical changes which it undergoes. The sputa may be milky white, green, brown, chocolate coloured, yellow, or obviously blood-stained. In the thick lower layer, pus corpuscles, elastic tissue, Dettrich's plugs, crystals of fatty acids, spirals, sarcinæ, and leptothrix, flagellated monads, staphylococcus pyogenes aureus, and the micro-organisms usually associated with putrefactive changes may be present.

The **physical signs** in cases of gangrene present no distinguishing features. They depend upon the condition of the affected area, whether it be consolidated, or softening, or already excavated, and upon the previous condition of the lung, if it has been affected by some definite lesion such as bronchiectasis.

In children the signs are often those of broncho-pneumonia only. The difficulties which attend the localisation of the exact site of the lesion, especially if a cavity has formed, are referred to later in the chapter on the Surgical Treatment of Pulmonary Cavities.

Diagnosis.—The presence of the condition is, as a rule, readily recognised by the characteristic odour of the breath and

P XI

BRONCHO-PNEUMONIA

(CATARRHAL PNEUMONIA. LOBULAR PNEUMONIA)

	PAGE
Etiology	252
Morbid anatomy	253
Associated lesions	255
Symptoms and course	255
Physical signs	256
Diagnosis	256
Prognosis	257
Treatment	257

THIS form of pneumonia, which until lately was considered to be generally due to the direct extension of a simple catarrhal inflammation from the bronchi to the lungs, is now by many writers ascribed to the presence of septic organisms, which multiply in the oro-nasal passages where they are commonly present, and owing to changes in the tracheal and bronchial mucous membrane are enabled to make their way thence towards the lungs, into the alveoli of which they either spread or are drawn by inhalation.

Various organisms are found in broncho-pneumonia, and as a rule infection is mixed. The micrococcus lanceolatus, the streptococcus pyogenes aureus and s. p. albus, staphylococci, and Friedländer's pneumococcus have all been identified.

Etiology.—In its typical form the affection is essentially one of infancy and childhood, and occurs with especial frequency as a secondary complication of bronchitis, whooping cough, and measles.

It is also often present in cases of diphtheria when the disease has extended to the bronchi, and it has been observed in association with extensive burns of the skin (Wilks). The pulmonary affection common in influenza resembles broncho-pneumonia in its distribution, although differing from it somewhat in its nature (*vide* p. 530).

A diffuse broncho-pneumonia may occur as a complication of bronchiectasis, and of chronic pneumonia. It may also be caused by the entrance of septic material into the bronchi. Malnutrition, rickets, the breathing of impure air, and any conditions leading to a defect of resisting power, act as powerful predisposing causes of the affection. It may occur as a secondary complication of collapse of the lungs, but more often is the direct cause of that condition.

The affection has the highest mortality in early childhood, a very large proportion of fatal cases occurring before the age of four years. It is also a very serious disease when it occurs as a complication of bronchitis in old people.

Morbid anatomy.—Both lungs are affected as a rule, but the lesions are generally more extensive in one than the other. The surface of the lung is usually studded with depressed areas of collapse presenting the purple colour and sharply defined angular outline typical of that condition. Elsewhere small areas of consolidation project from the surface, and the pleura covering them may show evidence of inflammation, although this as a rule is slight, and rarely approaches in extent to that observed in pneumonia. The lobules surrounding the solid areas are often over-distended with air.

On section, the lung is seen to be congested, and scattered through it there are rounded areas of consolidation about the size of peas, with a slightly granular surface and ill-defined outline. The inflamed lobules are in the early stage of a deep reddish-brown tint; at a later period they may become grey, and still later they are of a yellow or greyish-yellow colour. They may be discrete or aggregated in racemose groups, the confluence of which may produce large areas of consolidation involving in some cases the greater part of a lobe. Even then, however, there is little difficulty in deciding on the nature of the process, as the consolidated part lacks the uniform appearance of pneumonia, and here and there small areas of unaffected or collapsed or merely œdematous lung will be seen. An entire lobe is, moreover, rarely if ever completely consolidated.

Collapse may be present in scattered areas, or occur in patches of considerable extent. The latter are especially common towards the base of the lung. Inflamed lobules are often present in the collapsed patches, and may be recognised by their granular surface. On compression of the lung, beads of puriform mucus will be seen to issue from the smaller bronchi, which are acutely inflamed, whilst the lining membrane of the larger tubes is also inflamed and of a deep purple colour.

On microscopical examination [illegible] fig. 76) the alveoli are seen to be filled with small ro [illegible] and large round or oval cells of an epithelial type co [illegible] ne or more nuclei. The latter are the result of a proli [illegible] he alveolar epithelium, as is shown by their attachmer [illegible] the wall of the air vesicle, and by their greater abunda [illegible] iphery of the exudation. Fibrin may also be present, [illegible] less extent than in pneumonia. The cells of both varieties are often seen to be undergoing a process of fatty degeneration. The bronchioles of the inflamed areas are also filled with an exudation chiefly consisting of small round cells.

At the margin of the inflamed area, where the process is less advanced, the epithelial cells lining the alveoli are enlarged and undergoing proliferative changes; the walls are thickened and the vessels are distended with red blood corpuscles. The interlobular septa are also swollen, chiefly from congestion of the vessels with coloured corpuscles. The walls of the finer bronchi show infiltration with small round cells, and the epithelial lining is seen to be undergoing active proliferative changes, and may in places be detached. Peribronchial infiltration is commonly present. The smaller tubes are often dilated, this change being especially common when the disease has followed whooping cough. In cases due to the inhalation of septic material, micrococci in large numbers may be found in the inflamed alveoli and around the bronchi and vessels.

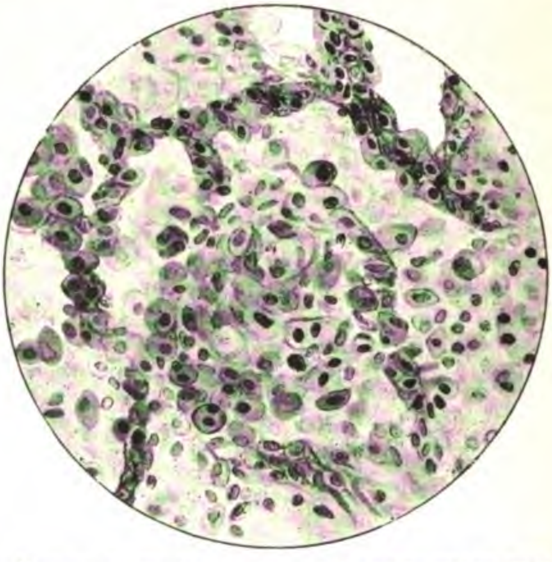


FIG. 76.—BRONCHO-PNEUMONIA, SHOWING AN ALVEOLUS FILLED WITH LARGE CATARRHAL CELLS AND THICKENING OF THE ALVEOLAR WALL

The consolidated areas may undergo a process of softening, small abscesses being formed and extensive destruction of the lung taking place. In such cases gangrene may follow. The diffuse broncho-pneumonia which often occurs at the termination of a case of bronchiectasis is an example of this process.

In some of the cases which recover, the inflammatory process undergoes arrest, and absorption of the effused products is followed by a return of the part to its normal condition. In other cases the lung remains in a condition of chronic pneumonia, and bronchiectasis subsequently occurs.

As stated in the chapter on Emphysema, a condition of acute over-distension of the pulmonary alveoli, the result of violent cough, is often found post mortem in cases of broncho-pneumonia following whooping cough in children, but the atrophic changes essential to true emphysema are absent. Emphysema, however, may have preceded the acute attack, or it may subsequently supervene in cases where resolution is incomplete and fibrosis of the lung follows.

Associated lesions.—The bronchial glands are almost invariably enlarged, but they are not caseous. The right auricle and ventricle are dilated as a result of the obstruction of the pulmonary circulation, and all the organs emptying their blood into the systemic veins show signs of congestion. The intestines often show evidence of the presence of acute catarrh, and sometimes even of ulceration. Thrombosis of the pulmonary artery and pericarditis may be mentioned as rare complications.

Symptoms and course.—The onset of the disease is marked by a sudden rise of temperature. A temperature ranging between 103° and 105°, or even higher, is by no means uncommon. The fever is of an irregular type, and considerable remissions and exacerbations occur, often without obvious cause. The difference between the morning and evening temperature may be two or three degrees or more, the higher temperature being occasionally recorded in the morning. Rigors rarely occur, and vomiting is uncommon. Cerebral symptoms, such as delirium and convulsions, suggestive of the presence of meningitis, may occur in children at the onset, but they are not very common, and are usually of short duration.

The breathing is markedly quickened, from 50 to 80 or even more short and shallow respirations per minute being often observed. There is an urgent sense of dyspnœa, the alæ nasi are widely dilated, and the patient prefers to lie with the head high and the back well supported by pillows.

When the disease occurs as a complication of bronchitis, its onset is often first recognised by a change in the cough, which, if it has previously been loose and painless, now becomes short, dry, more frequent, and accompanied by pain. Free secretion from the bronchial mucous membrane ceases, and the sputum is viscid and difficult to expel; it may be streaked with blood, but never presents the rusty character typical of pneumonia. Expectoration is usually absent in young children.

The face is at first pale, then, as the breathing becomes increasingly laboured, livid or cyanotic in tint. The lips and the finger-nails also show cyanosis. The expression is anxious, and restlessness is often marked. The pulse is small, feeble, and very quick—150 or more—and sometimes uncountable. Heat and dryness of the skin may alternate with perspiration. The tongue soon becomes dry; diarrhœa is a somewhat common symptom; and in acute cases emaciation rapidly occurs. The disease, if not quickly fatal, as it may be in infants and young children, usually runs a somewhat protracted course, from fourteen days to three weeks being a common duration, and the period may be much more prolonged. The temperature falls by lysis, a critical defervescence being very rarely observed. The dyspnœa slowly passes off, the pulse gradually diminishes in frequency, and the physical signs clear up.

Physical signs.—On inspection of the chest the inspiration is seen to be short and the expiration prolonged. There is some elevation but little true expansion of the chest, the lower intercostal spaces, the submammary regions, and the epigastrium receding with inspiration, whilst the sternum is raised and arched forward. The percussion note may be normal, if the consolidated areas are few in number, and widely scattered through the lungs. If a sufficiently extensive area has been consolidated by coalescence of foci of inflammation, and if, as is often the case, there is collapse of neighbouring lobules, resonance may be impaired, or there may be patches of dulness. At a more advanced stage a hyper-resonant note may be obtained owing to the over-distension of vesicles surrounding small foci of inflammation.

The breath sounds are usually harsh over the upper lobes, absent, feeble, or bronchial at the bases, their exact character being dependent upon the relative predominance and extent of consolidation and collapse. Collapse is indicated by retraction of the side and diminution or absence of breath sounds, and of vocal fremitus and vocal resonance; consolidation, of some extent, by an increased conduction of the voice sounds, and by the presence of bronchial breathing and bronchophony.

The typical adventitious sounds of broncho-pneumonia are fine crackling râles, not limited to the period of inspiration; rhonchus and sibilus, if present, are due to co-existing bronchitis.

The percussion note in collapse is less dull than in consolidation, and may have a tympanitic quality.

Diagnosis.—The diagnosis of broncho-pneumonia from *acute disseminated tuberculosis* of the caseous type may be very difficult, particularly if the case is seen at a late stage when marked emaciation has taken place, and, owing to the previous dilatation of the finer bronchi, the râles have acquired a coarse and almost metallic quality, highly suggestive of softening tubercle. It should be remembered that this form of tuberculosis is very rare, the acute miliary variety, which is unaccompanied by softening and marked by relatively few adventitious sounds, being far more common in children, in whom the difficulty of diagnosis is most likely to arise. The physical signs in tuberculosis will almost certainly be more marked in the upper lobes, whilst in broncho-pneumonia with bronchiectasis the lower lobes are, as a rule, more affected.

If expectoration is present, and the sputum contains tubercle bacilli, the difficulty at once disappears. Careful attention to the previous history of the patient and to the course of the illness will often settle the point; but, if doubt remains, it is well not to commit oneself to an opinion that tubercle is present.

In uncomplicated *collapse of the lungs* there is no rise of temperature. The physical signs of collapsed lung, and the way in which they differ from those of broncho-pneumonia, have been already stated.

From *bronchitis* the disease is differentiated by the higher range of temperature and the character of the adventitious sounds.

When the greater part of a lobe has become consolidated by the confluence of separate foci of broncho-pneumonia, it may be extremely difficult to diagnose the affection from *pneumonia* by the physical signs alone; but the history will be different in the two cases, and the temperature will, as a rule, show less marked remissions in pneumonia than in broncho-pneumonia. If the illness has lasted over a week and no crisis has occurred, the case is probably one of broncho-pneumonia.

Typhoid fever, especially in children, may be accompanied by extensive broncho-pneumonia at an early stage of the disease. In such cases the absence of any obvious cause of the pulmonary affection, such as previous bronchitis, and the presence of enlargement of the spleen point to a diagnosis of typhoid fever.

The cerebral symptoms that often accompany the onset of broncho-pneumonia in children may at first suggest *meningitis*, but the fact that many acute diseases are ushered in by similar symptoms should prevent a rash diagnosis of meningitis, and the presence of a considerable degree of dyspnœa and of the physical signs of pulmonary disease are strongly in favour of that of broncho-pneumonia.

Prognosis.—This is grave in all severe cases, as the disease has a high rate of mortality. In infants and in young rickety children the probability of a fatal termination is always great, especially when the affection complicates whooping cough. The association of extensive collapse and bronchitis affecting the finer tubes with cyanosis or lividity, constitutes a very unfavourable condition.

A very high temperature is always an unfavourable sign.

The character of the pulse is often a safer guide than the rate of the breathing, a very feeble, rapid, and 'running' pulse being of more serious import than rapid respiration. Convulsions occurring at the onset of the disease are not necessarily unfavourable, but at a later period they are very likely to be followed by death. Broncho-pneumonia may lead to enlargement of the bronchial glands, which may be a source of trouble at a later period, and to bronchiectasis. Children weakened by broncho-pneumonia are very liable to infection from tuberculosis.

Treatment.—The general treatment of broncho-pneumonia, whether in the young or old, should be stimulant, and such as is likely to support the patient's strength.

The room should be maintained at an even temperature of 65° F., and the air should be moistened by the use of a bronchitis kettle. It is usual to surround the bed of the patient, if a young child, with a tent; but opinions differ as to the value of this measure, and it is very easy to produce within the tent an atmosphere so saturated with watery vapour that respiration is impeded rather than assisted. If it is used, great care is necessary to maintain the temperature of the room in the early hours of the morning, or the patient may be chilled by his cold and damp surroundings.

Stimulant expectorants, such as carbonate of ammonia and senega, may be required from the outset.

Dr. Burney Yeo speaks highly of the use of benzoate of soda internally, and of a warm alkaline spray containing bicarbonate of soda and glycerine of carbolic acid. The effect of alkaline remedies in diminishing the viscidity of the bronchial secretion has been referred to in discussing the treatment of bronchitis. This mode of treatment has also a beneficial effect upon the cough by relieving the dryness of the bronchial mucous membrane. If the cough is very troublesome in children, small doses of Dover's powder (gr. $\frac{1}{2}$–1) may be given twice daily, but, speaking generally, the administration of opiates is to be avoided in this disease as much as in bronchitis.

As a rule, all lowering remedies should be avoided, except in the early stage in children, when very small doses of tincture of aconite (♏$\frac{1}{4}$ to ♏1 every hour) may be given for a few hours. Its use sometimes induces diaphoresis, and so lowers the temperature and relieves restlessness. Also at a later period, when the bronchi tend to become obstructed and there is much cyanosis, an emetic of ipecacuanha may be useful by effecting the expulsion of the secretion during the act of vomiting.

The early use of oxygen inhalations is often of great service by improving the action of the heart. The oxygen should be warmed by passing it through a spiral metal coil placed in a vessel containing hot water; if this is done the inhalation may be continuous. Subcutaneous injections of strychnine (gr. $\frac{1}{100}$–$\frac{1}{50}$) at intervals may be employed as aids to other methods of treatment; the drug acts by stimulating the respiratory centre. Stimulants are often necessary from an early stage of the illness, and brandy may be given in doses of from 10 to 15 minims, or more, every two hours to an infant, and the quantity may be gradually increased if the symptoms become more severe. In some cases, large quantities, such as ℥iss in the twenty-four hours, may be given to an infant. The frequency of the pulse and respirations constitutes the best guide to the necessity for the use of stimulants, and also for an increase of the dose.

Dr. Wilson Fox writes with approval of the use of cold baths and cold applications to the chest in broncho-pneumonia in children. The cold bath, at a temperature of 65° to 70° F., is used for a few minutes (one to three).

Jurgensen preferred a longer immersion (twenty minutes) in a bath at a temperature of from 77° to 80° F. Cold sponging of the chest may also be employed for the reduction of temperature, and is preferable to the use of cold cloths. It may be repeated as often as the threatening symptoms return, or the temperature rises to 103° F. A diminished frequency both of the pulse and respiration is the best evidence that the application of cold is doing good, whatever method may be employed.

A valuable method of treatment when the temperature is high and death is threatening from engorgement of the venous system, as evidenced by cyanosis and urgent dyspnœa, is to place the child in a hot bath (temp. 104° to 110° F.), and at the same time to pour cold water (temp. 60° to 65° F.) over the chest and back. The effect is to produce a sudden deep inspiration, in which collapsed lobules are doubtless opened up and air passes beyond retained mucus, which it subsequently helps to expel. The breathing for a time continues deeper, and cyanosis may be markedly diminished.

As local applications to the chest poultices, at one time almost invariably used, are now somewhat out of fashion. To surround the chest of a rickety child with a heavy poultice must impede the expansion and favour collapse of the lungs; but this objection is to some extent removed by applying the poultice only to the back of the chest, whilst the front is covered with cotton wool, on which turpentine or dry mustard may be sprinkled. Mustard leaves are also of great use as a local application. The choice of the local application will depend very much on the efficiency of the nursing; in the absence of trained attendants the use of poultices should, as a rule, be avoided.

The food should consist of milk, jelly, beef-tea, chicken broth, essences and juices of meat, and eggs in milk. It should be given at intervals of from two to three hours in severe cases.

When the symptoms are subsiding especial care is necessary to prevent the occurrence of a relapse, and, during convalescence, far greater watchfulness is required than after recovery from pneumonia. Cod-liver oil and tonics are then of great service.

J. K. F.

CHAPTER XXII

PULMONARY FIBROSIS

CHRONIC PNEUMONIA, CIRRHOSIS OF THE LUNG, INTERSTITIAL PNEUMONIA

	PAGE
Etiology	260
Morbid anatomy	261
Symptoms	264
Physical signs	264
Course	265
Diagnosis	265
Prognosis	266
Treatment	266
Reticular fibrosis	266

THE essential feature of the condition to which the above terms are applied is the presence of an excess of fibrous tissue within the lung. This may arise from a development of new tissue in the walls of the pulmonary alveoli or from fibrous thickening of the sub-pleural, interalveolar, interlobular, peribronchial, or perivascular connective tissue. In the majority of cases the above lesions are found in association, but one or other may predominate.

Etiology.—Fibrous induration of the lung may occur as a result of a great variety of conditions.

It may be secondary to—

1. *Pulmonary tuberculosis.*—This is by far the most common cause, the change being especially marked in the chronic and fibroid forms of the affection.

It will be more convenient to consider here only those forms of the lesion which occur independently of the presence of tubercle.

2. *Pneumonia and subacute indurative pneumonia* (lobar type).—The occurrence of induration as a sequel of pneumonia and in the special form of disease to which the latter of the above titles is given is dealt with in the preceding chapter.

3. *Broncho-pneumonia with collapse.*—In children this affection not infrequently passes into a chronic condition accompanied by

induration of the lung, which commences as a fibrous thickening of the alveolar walls.

4. *Bronchiectasis.*—As a rule, when dilatation of the bronchi is found in association with induration of the lung, the pulmonary lesion is primary; bronchiectasis, from whatever cause arising, may however be followed by induration of the lung. Usually the change in such cases is limited to the neighbourhood of the dilated tubes.

5. *The inhalation of irritant substances.*—In the large majority of cases of so-called 'knife-grinders' phthisis' or 'stonemasons' phthisis,' &c., the condition is the combined result of tuberculosis and fibrosis of the lung, the latter being only in part due to the irritant effect of solid particles inhaled in the course of the patient's occupation. In a certain number, however, tubercle is absent, and in such the induration is to be ascribed to the inhalation of irritant substances (*vide* 'Diseases due to Inhalation of Dust,' p. 269).

6. *Pleurisy.*—Lesions of the pleura may be the starting point of indurative changes in the lung, a condition to which the term 'pleurogenic cirrhosis' is applied. We are of opinion that such a sequence of events is not of such common occurrence as it appears to be considered by some writers; it is indeed in many cases remarkable to what a short distance the lesions of subjacent parts of the lungs extend in cases of pleurisy, whether acute or chronic, and we doubt whether pleurisy unaccompanied by any pulmonary lesion is capable of producing extensive fibrosis of the lung.

7. *Syphilis.*—On reference to the chapter on Pulmonary Syphilis (p. 431) it will be seen that the lungs of infants suffering from congenital syphilis often present in a very typical manner the lesions characteristic of interstitial pneumonia. In adults, areas of fibrous induration occasionally mark the site of previously existing gummata.

8. *Foreign bodies in the bronchi.*—Induration of the lung in association with bronchiectasis and other lesions is not infrequently the result of the long continued presence of foreign bodies in the air passages.

9. The pressure of an *aneurysm* may induce localised fibrosis of the lung.

10. Malignant growths of the lung, pleura, or œsophagus may also set up indurative changes in the lung.

Morbid Anatomy.—Three varieties of the lesion are met with: *a.* The *massive* or lobar form; *b.* The *insular* or broncho-pneumonic form, in which fibrosis occurs in patches; *c. Reticular fibrosis*; this variety is separately considered at the end of this chapter.

a. The lobar form, as a rule, affects one lobe of a lung. It is unilateral in distribution, and involves the lower lobe. The lung is of a greyish colour, with irregular marbled areas, solid and indurated on section; in some cases it presents a finely granular appearance. It subsequently shrinks, owing to the contraction of the newly-formed fibrous tissue. Dilatation of the bronchi, generally of the cylindrical variety, but sometimes of the saccular, may occur at a later period, and the changes incidental to that affection, viz. diffuse septic broncho-pneumonia, necrosis, and gangrene are common in fatal cases.

Bronchiectasis is, however, more common in the succeeding variety.

Pulmonary aneurysms may form in cavities. Emphysema is usually present in the neighbourhood of the contracting lesions, and pleural adhesions are commonly found.

b. The broncho-pneumonic form, which is the most common, is often secondary to measles, whooping cough, and scarlet fever.

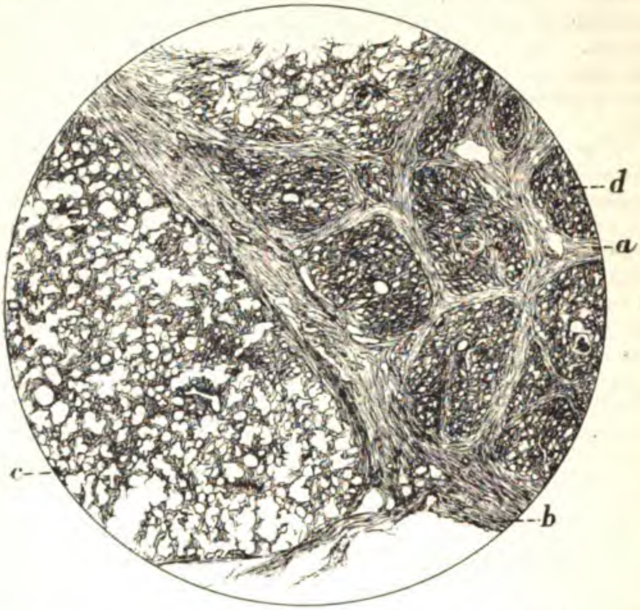


FIG. 77.—PULMONARY FIBROSIS (× 7 diameters)

Showing, *a*, thickened interlobular septa ; *b*, thickened pleura ; *c*, emphysematous lung in neighbourhood of lesion ; *d*, lobules with dilated bronchiole in the centre, alveolar walls and connective tissue structures much thickened.

The fibrosis affects certain areas of the lung, which are usually deeply pigmented, and may be separated from one another by tissue, which may be either normal or emphysematous or traversed by fibrous bands.

The lesions may be situated in any part, but are more commonly present in the lower lobes. Sacculated bronchiectasis often occurs. If excavation occurs, aneurysms, usually of small size, may form on branches of the pulmonary artery, and profuse and fatal hæmoptysis may follow rupture of one of them into a neighbouring cavity.

Microscopical appearances.—In pulmonary fibrosis of the lobar and broncho-pneumonic forms a new growth of connective tissue is found within the alveoli, and in the connective tissue framework of the lung.

The accompanying drawing (fig. 77) of a section of the lung from a case of chronic pneumonia and bronchiectasis in a child, the subject of congenital syphilis, illustrates these changes.

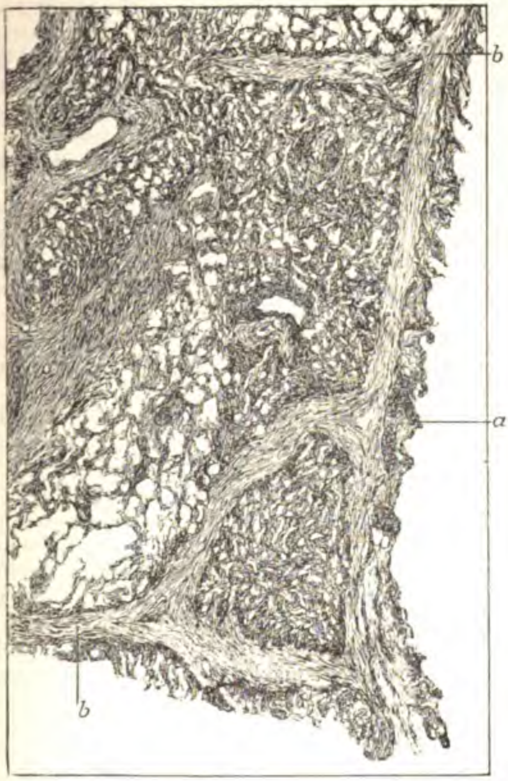


FIG. 78.—ACUTE PLEURISY, WITH THICKENING OF THE CONNECTIVE TISSUE OF THE LUNG (× 7 diameters)

a, fibrinous exudation on the surface of the pleura; *b*, thickened septa.

The alveolar walls are thickened by a growth of fibro-cellular
e, chiefly consisting of long fusiform cells, and the vesicles are
ressed or obliterated. They usually contain large epithelial
and leucocytes, and sometimes granular corpuscles and fat
ules. The connective tissue throughout the affected area shows
mmatory changes, and the thickening of the interalveolar and
lobular tissue may be more obvious than the changes in the

alveolar walls or within the alveoli. The smaller bronchi are usually dilated and show the lesions incidental to chronic inflammation; fibrous thickening of the peribronchial tissue is also commonly present.

The pleura overlying the indurated lung is almost invariably thickened, the extent of this change depending upon the amount of contraction which has taken place within the lung. In very chronic cases the pleura may be of almost cartilaginous density. The pathology of this lesion is discussed in the chapter on Chronic Pleurisy (*vide* p. 578).

The accompanying drawing (fig. 78) is a section of the pleura and the surface of the lung taken from a case of acute pleurisy. It shows that thickening of the interlobular septa *may* occur as a result of inflammation of the pleura; but the change is, as a rule, limited to the subjacent part of the lung, and, independently of pneumonia or chronic bronchitis, cannot be considered as *per se* a cause of fibrous overgrowth of the alveolar walls.

Symptoms.—If the case is observed from the onset and watched for a long period, the symptoms will depend first upon the nature of the primary disease, and later upon the subsequent changes in the lung.

Careful observation shows that the presence or absence of bronchial dilatation exercises the most important influence both on the symptoms and prognosis of the disease.

The cough which is always associated with bronchial dilatation induces emphysema, the putrid secretions are inspired into other parts of the lung, bronchitis becomes general, and the other characteristic symptoms of bronchiectasis become manifest.

If, however, the affected lung is much shrunken and the bronchi are obliterated, there will be more marked contraction of the side, great displacement of the mediastinum, heart, diaphragm, and abdominal organs. A moderate degree of bronchitis with cough and expectoration, and dyspnœa on exertion, are the symptoms commonly present. Such a condition is quite compatible with fair health, and the absence, for considerable periods at a time, of pyrexia and any very urgent symptoms.

The **physical signs** observed in cases of fibrosis, associated with retraction of the lung and bronchiectasis, are absence of expansion, increase of the vocal fremitus, the presence of bronchophony or pectoriloquy and of a percussion note, which is either dull or of wooden or tubular or amphoric quality, according to the size of the cavities and their nearness to the surface of the lung. The breath sounds are either bronchial or cavernous, and large bubbling, gurgling, or metallic râles are audible. Signs of bronchitis may be present on the affected side in the neighbourhood of the area of induration or in the opposite lung.

If the lung is retracted and the bronchi obliterated, such of the above signs as are dependent upon the presence of dilated tubes will be wanting. The respiratory sounds over the contracted lung may in such cases be faintly bronchial in character.

The unaffected lung may have undergone compensatory enlargement, as shown by the presence of puerile breathing and a hyper-resonant percussion note, which latter may extend considerably beyond the mid-sternal line.

The direction of the cardiac displacement will depend upon the site of the pulmonary lesion. The impulse in cases of extreme contraction of the left lower lobe may be felt in the posterior axillary line, and in a case recently under our care it was obvious when the patient was looked at from behind.

The **course** of the affection is in all cases chronic, its duration being generally reckoned by years rather than months.

As the case progresses towards a fatal termination dropsy is very likely to occur, it is usually limited to the lower extremities, and is due to some degree of cardiac failure. Albuminuria and amyloid disease may also be present. The final stage of bronchiectasis has already been several times referred to.

Diagnosis.—The condition is distinguished from pulmonary tuberculosis of the very chronic type primarily by the absence of bacilli from the sputa, and also by the fact that, unlike tubercular disease, it is usually unilateral, and does not extend from the upper to the lower lobe in the manner characteristic of the great majority of cases of that disease. Primary basic tuberculosis accompanied by fibrosis is a rare condition which may closely simulate chronic pneumonia; the differential diagnosis depends again mainly upon the results of the examination of the sputa. Tuberculosis may, however, supervene upon fibrosis which was originally non-tubercular.

Difficulty in diagnosis may arise in cases in which the imperfect absorption of a pleural effusion has been followed neither by expansion of the lung nor by dilatation of the bronchi. This is characterised by a more localised retraction of the chest wall than is met with in pneumonia, with diminished vocal fremitus and feeble breathing, or by an absence of respiratory and adventitious sounds. If the presence of fluid is suspected it is now the almost invariable practice to insert an aspirator needle and to be guided by the results obtained.

Secondary bronchiectasis in a lung which has been collapsed by the pressure of pleural effusion gives rise to a condition so closely resembling that under consideration as to be almost indistinguishable from it by physical signs. The history of the acute illness with which the symptoms commenced may suffice to decide the point.

Malignant growths in the lung and mediastinum are occasionally accompanied by physical signs suggestive of chronic pneumonia, particularly when, owing to compression of a main bronchus, the tubes throughout the lung have undergone dilatation. In a case of the kind recently under the care of the writer, the diagnosis of neral sacculated bronchiectasis was verified post mortem; but the esence of the growth, which nowhere approached the surface of e lung, had not been suspected.

The presence of pressure signs and of dulness transgressing the median line, pain in the chest, and currant jelly-like expectoration point to a diagnosis of malignant growth. Extreme cardiac displacement, compensatory enlargement of the opposite lung, and a duration more prolonged than two years suggest that the case is one of chronic pneumonia.

The diagnosis between chronic pneumonia with bronchiectasis, and the perforation of the lung by an empyema or hepatic abscess often presents great difficulties.

The decision in most cases of the kind depends upon the results of an exploratory puncture with an aspirator needle.

Prognosis.—The duration of the disease is, as a rule, prolonged, and may extend to ten or twenty years or more.

The prognosis is decidedly most favourable in cases in which the bronchi are not dilated.

Death is usually preceded either by cardiac failure and dropsy or by septic broncho-pneumonia and gangrene.

Treatment.—It is important to maintain the strength and nutrition by a liberal diet, with tonics and cod-liver oil. The subjects of pulmonary fibrosis are very liable to bronchial catarrh, and should be protected from damp and cold by warm clothing, and when possible by a residence during the winter in a dry and warm climate. Failing this, in severe cases they are usually better indoors during that season unless the weather is exceptionally fine and bright.

Profuse and fœtid expectoration should be treated according to the methods recommended in the chapter on Bronchiectasis.

It is unnecessary to describe in detail the treatment suitable for the various complications of pulmonary fibrosis.

c. RETICULAR FIBROSIS.—This form of fibrosis is of purely pathological interest, as no case has, so far as we are aware, ever been recognised during life. A typical case is reported by Dr. Percy Kidd,[1] and another, of a somewhat similar character, by Dr. W. McCollom.[2]

The writer is indebted to Dr. Percy Kidd for kindly lending the microscopical specimen obtained from the case above referred to figured on the next page.

The lungs in this case presented the following appearances. They were spongy but firm; emphysema was present at the margins; the pleuræ were opaque, slightly thickened, especially along the posterior borders. On section they were rather dry, of a deep red colour, and traversed throughout by numerous intersecting greyish fibrous strands, which seemed in places to follow the lines of the interlobular septa. These fibrous bands were scattered symmetrically throughout both lungs, but varied somewhat in thickness and number in different parts. The interlobar septa were not thickened. A small mortar-like mass, embedded in pigmented indurated tissue, was situated in the left upper lobe, and two firm encapsulated subpleural caseous nodules in the right lower lobe. No miliary tubercles and no tubercle bacilli were found. The bronchi were slightly dilated in places.

---

[1] Soc. *Trans.* vol. xxxvii. p. 126.

Microscopical examination.—There was marked fibro-cellular thickening of the outer coat of bronchi and bronchioles, involving also the adjacent arteries and veins.

The mucous membrane in most of the bronchioles was atrophied; in others it was thickened and fibroid.

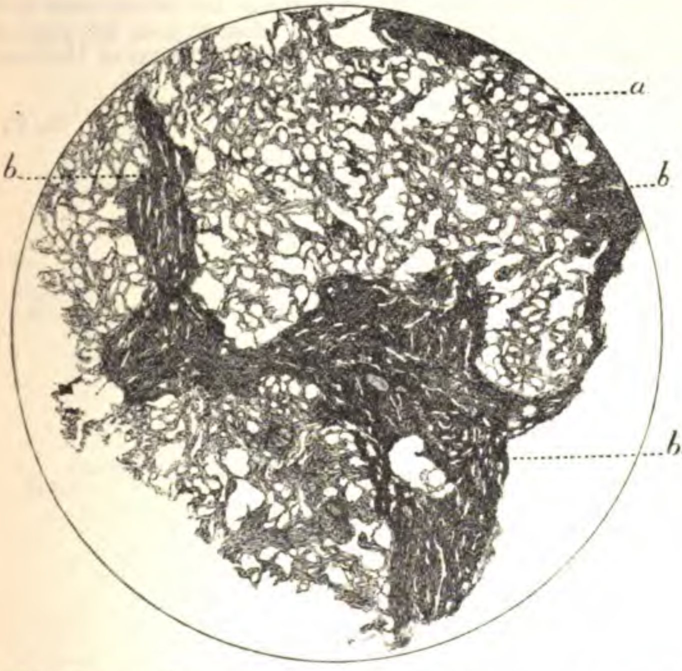


Fig. 79.—Reticular Fibrosis of the Lungs, with Emphysema (× 7 diameters)

a, dilated vesicles with thickened alveolar walls: *b*, pigmented fibrous bands traversing the lung.

Peribronchial thickening was the prominent lesion; it varied from a purely cellular infiltration to a dense fibrous growth. Emphysema was most marked in the neighbourhood of the diseased bronchi. The fibrous bands contained numerous capillary-like vessels.

The patient was a woman æt. 37, who had enjoyed good th up to twelve months before her admission to hospital. illness began with retching and vomiting, followed by cough, thick yellow expectoration, and shortness of breath on exertion.

She had had slight hæmoptysis, and more recently had suffered from palpitation. Death occurred eleven days after admission from anasarca and gradually increasing dyspnœa.

Dr. Kidd discusses the origin of the lesions, and considers the evidence insufficient to allow of their being ascribed either to syphilis or tuberculosis. He regards the affection as due to a progressive chronic bronchitis and peribronchitis, leading to fibrosis of the lungs.

A case of a similar kind reported by Dr. Perry[1] is described in the chapter on Pulmonary Syphilis (*vide* p. 439).

The writer also desires to acknowledge his indebtedness to Dr. Percy Kidd for the classification here adopted, and for other information derived from his paper on Fibroid Disease of the Lungs (*vide* 'Clinical Journal,' Nov. 7, 1894).

J. K. F.

---

[1] *Path. Soc. Trans.* xlii. p. 53.

CHAPTER XXIII

DISEASES DUE TO THE INHALATION OF DUST

(PNEUMOCONIOSIS, ANTHRACOSIS, SIDEROSIS)

PAGE
Pathology 269
Morbid anatomy 270
  Changes in the bronchi 270
  Emphysema 270
  Fibrosis 270
  Pigmentation 271
  Excavation 271
  Bronchial glands 271
  Pleura 272
Etiology 272
  Varieties of dust 272
    (*a*) **Silica and alumina** 272
    (*b*) **Carbon** 273
    (*c*) **Peroxide of iron** 273
    (*d*) **Organic materials** 273
Symptoms 273
Course 274
Physical signs 275
Treatment 275

It has long been known that employment in certain occupations **involves a** special liability to a slowly progressive disease of the **lungs**, but opinion has been greatly divided as to its precise nature.

Pathology.—The earlier view that under such circumstances **extensive** induration and destructive changes, particularly the formation of cavities, may occur independently of the presence of **tubercle** is no longer tenable in the face of the repeated discovery **of tubercle** bacilli in the sputa of patients suffering from the **affections** generally termed 'knife-grinders' phthisis,' 'grinders' **rot,**' '**stone**-grinders' phthisis,' 'potters' phthisis,' &c.

As already stated in the chapter on Pulmonary Fibrosis, we consider that the lesions of the above nature are in great part of tubercular origin. Such cases, indeed, constitute some of the most typical examples of the fibroid variety of pulmonary tuberculosis (*vide* p. 342).

It is possible, however, that in some cases of the kind which prove fatal from pulmonary tuberculosis the early lesions may not have been of that nature, but on this point it is difficult to speak with certainty. The museum of the Middlesex Hospital contains a series of preparations presented by the late Dr. Greenhow, whose work in connection with this subject is well known,[1] illustrating in a very complete manner the morbid changes incidental to employment in various trades.

Morbid anatomy.—The chief pathological lesions found are:—

1. *Changes in the bronchi.*—The first effect of the constant presence of spiculated and angular particles of metal or stone in the inspired air is to produce irritation and catarrhal inflammation of the laryngeal and bronchial mucous membrane. The process subsequently becomes chronic; thickening of the bronchial walls and fibrous changes in the peribronchial tissues occur, and are followed in many cases by bronchiectasis. In areas of induration the latter change is generally well marked, but more rarely the bronchi are found to be obliterated there. Bronchiectasis, when widely distributed, is usually of the sacculated variety. The dilated tubes may contain deeply pigmented puriform material.

2. *Emphysema* is commonly present, and is either general and of the 'large-lunged' variety or limited to the neighbourhood of chronic indurative lesions. It is the result of the cough which constitutes one of the chief symptoms of the affection.

3. *Fibrosis.*—This is the most characteristic lesion. It may be unassociated with tubercular changes, although the two are more often associated. Small scattered pigmented nodules are usually tubercles which have undergone fibrosis. By the aggregation of such nodules some at least of the larger areas of induration are formed. These may be found throughout both lungs, or the change may be limited to one lobe, or to the upper lobe and adjacent part of the lower lobe. The latter distribution of the lesions is common in the tubercular varieties of the affection, and is an important distinguishing feature of such cases. The fibrous areas are densely indurated. In one of Dr. Greenhow's specimens they are described as presenting 'a smooth section, not unlike pieces of indiarubber.' Fibrous overgrowth is also present at the margins of the more completely consolidated areas, and bands of pigmented fibroid tissue may traverse the lungs in various directions. The change, when not tubercular, is due to a chronic inflammatory process leading to the development of new tissue in the walls of the alveoli and in the connective tissue of the lung.

---

[1] *Third Report of Med. Off. to Privy Council*, 1861; and *Path. Soc. Trans.* vols. xvi. xvii. xx. xxi.

4. *Pigmentation* is hardly less characteristic of the condition than fibrosis. The colour varies to a slight degree only with the nature of the occupation followed. In the great majority it is deep black, this being especially marked in the lungs of coal-miners and others, in which particles of carbon are present in the inhaled dust. In a specimen of Dr. Greenhow's taken from a coal-miner, it is stated that 'when fresh, the lung on section exuded large quantities of thick, perfectly black fluid.' The lungs of iron-workers may be of a brownish-red colour, those of workers in ultramarine of a violet tint. The pigmentation may be present throughout the lung, and is almost invariably most marked in the areas of induration. It may be uniform or occur as speckles and streaks, and pigmented areas may be intersected by white lines. It was at one time doubted whether the inhaled dust is really the source of the pigment, but the proof of the presence of carbon in the black lungs of coal-miners, of oxide of iron in the red lungs of workers in that substance, and of solid particles of wood charcoal in the black lungs of a man engaged as a carman in the charcoal trade has finally settled a long disputed question.

5. *Excavation.*—Irregular cavities with black, ragged walls may be produced by the necrotic softening of consolidated areas. They are more often found in the upper lobes than elsewhere, and may be of very large size. Where such cavities are present, tubercular lesions are usually associated with fibrosis, induration, or calcareous degeneration. In a case reported by Dr. Greenhow,[1] which was fatal from hæmoptysis, a cavity in the left lower lobe contained dark fluid blood.

On microscopical examination the alveolar walls are seen to be thickened, and to contain masses of granules and of black pigment. Similar deposits are found around the bronchioles, and to a less extent throughout the lymphatic and connective tissue systems of the lung. The alveoli are distorted, owing to contraction of the newly-formed connective tissue, and, whilst some are obliterated or condensed, others show emphysematous enlargement. The contents of the alveoli consist of large catarrhal cells containing black pigment and free granules of the same material. The leucocytes in the pulmonary lymphatics appear to act as carriers of these particles of pigment. Pigmented fibrous bands may be found intersecting the lung, resulting chiefly from thickening of the interlobular connective tissue.

The hard nodules present the microscopical appearances of tubercles which have undergone fibroid transformation.

The *bronchial glands* are often somewhat enlarged, and almost invariably deeply pigmented and of a coal-black colour. The change is not confined to the large glands about the bifurcation of the trachea and around the main bronchi, but affects those which lie beside the tubes as they pass into the lung. This latter appearance, however, is constantly observed in cases of chronic the changes in the lung are limited to the lesions of fibrosis, as the variety of tuberculosis in which the granulations undergo fibroid transformation is characterised by a very similar course (*vide* p. 344). The presence of tubercle could at this stage hardly be discovered by physical examination; and the absence of bacilli from the sputum would not exclude it. It is in fact extremely probable that the areas of pigmentation and fibrosis, which at a later period may undergo softening, are formed at this period as a result of a chronic tubercular process. At a later stage, probably when excavation is in progress, the sputa generally, but apparently not invariably, present the characteristic appearance to which the name 'black spit' has been given. The following clinical history is attached to one of Dr. Greenhow's specimens: 'From a man aged 65 who had worked in a coal-mine from boyhood. He had been incapacitated from work during two years, during which time he had suffered from cough and shortness of breath, and had lost flesh. Ten days before his death he began to spit up large quantities of sputum resembling "black paint." *Post mortem.*—In the upper lobe there was an irregular cavity with ragged black walls adjacent to an area which was pigmented and indurated.'

---

[1] *Path. Soc. Trans.* vol. xvii. p. 24.

Pigmentation may certainly be present without causing black expectoration, and the 'black spit' may disappear and reappear as the process of disintegration of the lung is less or more acute. It has often been observed to appear long after the patient has ceased to work in the occupation which gave rise to the pulmonary disease. The quantity may be very large, as much as half a pint or more daily.

Gritty, black, and stony masses have been found in the sputa of grinders and masons.

If bronchiectasis is present the sputa will probably be fœtid and present the other characters peculiar to that affection.

Course.—Cases which have run a very prolonged course may terminate with very acute symptoms, as in the following, in which the lungs after death were deeply pigmented and presented areas of induration and of excavation. The case was that of 'a man aged 38, who had formerly worked as a French millstone maker, but for the last eight years as a stonemason. He had suffered from chronic cough, worse in the winter, for twenty years. About ten weeks before his death he caught cold, and was attacked by symptoms of rapid phthisis: diarrhœa supervened, and he ultimately succumbed from a severe attack of hæmoptysis.'[1]

As in fibroid tuberculosis the course of the disease may be apyrexial for long periods, but with the onset of softening the temperature assumes the remittent type characteristic of active tubercular disease of the lungs.

Emaciation may be rapid towards the close and may be accompanied by night sweating, diarrhœa, and dropsy.

---

[1] Catalogue of Middlesex Hospital Museum, specimen No. 1276. Reported by Dr. Greenhow, *Path. Soc. Trans.* vol. xvii. p. 24.

Physical signs.—The results of physical examination of the chest vary with the degree of emphysema present. As in fibroid tuberculosis the signs of consolidation may be masked by the condition of the surrounding lung, and similarly the adventitious sounds of bronchitis may obscure fine râles indicative of tubercular infiltration, or the larger râles which accompany its disintegration. If emphysema is absent, consolidation, softening, and excavation will be recognised by the ordinary signs which accompany those conditions.

Treatment.—If the patient is able, as soon as the symptoms appear, to give up his unhealthy occupation, the disease may be arrested; but, having generally been brought up to the trade and knowing no other, he rarely does so, and the disease usually carries him off before he has passed middle life.

The general plan of treatment suitable for such cases is that described under Pulmonary Tuberculosis. The disease, so far as it is directly due to the inhaled dust, may be prevented by the use of a cotton-wool respirator. It is true that workmen dislike it, but if certain privileges are accorded by manufacturers only to those of their employés who habitually wear the respirator, the objection to its use is apt quickly to disappear. The tubercular element in the disease is to be combated by attention to the hygiene of mines and workrooms, and by removal of dust from the atmosphere, which, so to speak, prepares the soil for the seed; by medical examination of the employés, and generally by measures which develop the resisting power of the individual and prevent tubercular infection. These latter are considered in detail in the chapter on Pulmonary Tuberculosis (*vide* p. 385).

CHAPTER XXIV

CONGESTION, ŒDEMA, AND HYPOSTATIC PNEUMONIA

	PAGE
Etiology	276
Morbid anatomy	279
Symptoms	280
Diagnosis	281
Treatment	281

THE common use, both by the medical profession and the laity, of the term 'Congestion of the Lungs' would appear to imply the existence of a clearly defined affection of that nature, whereas, except as a necessary accompaniment or sequence of certain well-understood morbid conditions, it is one of which the pathology is very obscure.

It is not easy to obtain a clear idea of what is meant by the term Congestion of the Lungs. The lung stands upon a different footing with regard to its vascular supply from that of any other part of the body, except the liver. It contains two systems of vessels almost distinct from one another: one the bronchial system, which subserves the nutriment of the tissues of the lung, and is part of the systemic system; and the other the pulmonary, which conveys the whole mass of the blood through the lungs in order that it may be modified *in transitu*.

There is no reason to suppose that the bronchial vessels differ in any way from those of any other part of the systemic circulation, either in regard to the degree to which they are governed by impulses from the vaso-motor centres in the medulla, or elsewhere, conveyed to them through the sympathetic nerves, or in regard to the part which they play in the processes of ordinary inflammation. At the same time we do not know of any experiments bearing upon the subject. But it is clear that if the smaller bronchial arteries were dilated, as are other vessels in the course of inflammation elsewhere, there would be an increase of arterial blood in the part. It is also clear that anything which causes obstruction to the general circulation would cause congestion of the bronchial vessels and lead *pro tanto* to an increase of improperly oxygenated blood in the lungs.

The pulmonary vessels are, however, upon a totally different footing.

It has long been discussed by physiologists whether or not the pulmonary arteries are governed by the sympathetic nerves. A series of important observations by Dr. J. R. Bradford seems almost to have settled the question in the affirmative; but even now, although there is no question about the facts observed, the interpretation of them cannot be said to be altogether beyond dispute.

The line of his argument is as follows :

1. Stimulation of the vaso-motor centre in the medulla increases blood pressure both in the aorta and the pulmonary artery.

The problem is to eliminate the effect of the increased pressure in the systemic circulation upon that of the pulmonary circulation.

2. Direct pressure on the aorta does not appreciably raise the blood pressure in the pulmonary artery.

The increased pressure in the aorta on stimulating the vaso-motor centre in the medulla is practically found to be produced almost entirely through the medium of the vessels supplying the abdominal viscera.

3. Stimulation of the vaso-motor centre in the medulla after section of the cord at the level of the seventh dorsal nerves (which is above the level at which most of the branches going to the abdominal viscera come off) does not increase the blood pressure in the aorta, but does increase it in the pulmonary artery.

4. Stimulation of the lower end of the divided cord increases the pressure in the aorta, but does not produce any influence upon the blood pressure in the pulmonary artery.

Dr. Bradford allows a possible fallacy in the argument, depending upon the question of the hepatic condition which he has not yet had the opportunity of clearing up.

It must therefore be held to be almost certain that the pulmonary arterioles may be dilated under the influence of the vaso-motor centres.

It is also clear that anything that causes obstruction on the left side of the heart, such as incompetence or stenosis of the mitral or aortic valves ; and anything which obstructs the general circulation, such as the circulation in the blood of certain poisons—amongst which carbonic acid must be included—will cause a passive congestion of the pulmonary vessels.

Whether it is advisable to use the terms arterial congestion and venous congestion of the lungs at all may well be open to doubt, considering the great complexity of the subject. But the reader will not fail to observe that, if there is such a thing as ‘arterial congestion,’ or ‘acute hyperæmia’ of the pulmonary vessels, the colour of the lung should be darker than before. Our own opinion is that the colour of the lung, as seen post mortem, depends not so ch on the part of the pulmonary system which is instrumental bringing the congestion or hyperæmia about, as upon the general condition of the blood as regards aeration at the time of death.

Two varieties of pulmonary congestion may be recognised—the one acute, the other chronic.

Acute congestion constitutes the first stage of pneumonia, and in a minor degree is present in all acute inflammatory affections of the lungs, whether simple or tubercular. It also occurs to some extent in rapidly developed cardiac failure, whether from myocarditis such as may occur in acute disease, or from some temporary condition in chronic valvular disease.

Acute congestion of the lungs may also occur during violent exertion and excitement, and possibly as a result of vaso-motor paresis, a condition which some writers are disposed to consider of much importance in the causation of hæmoptysis.

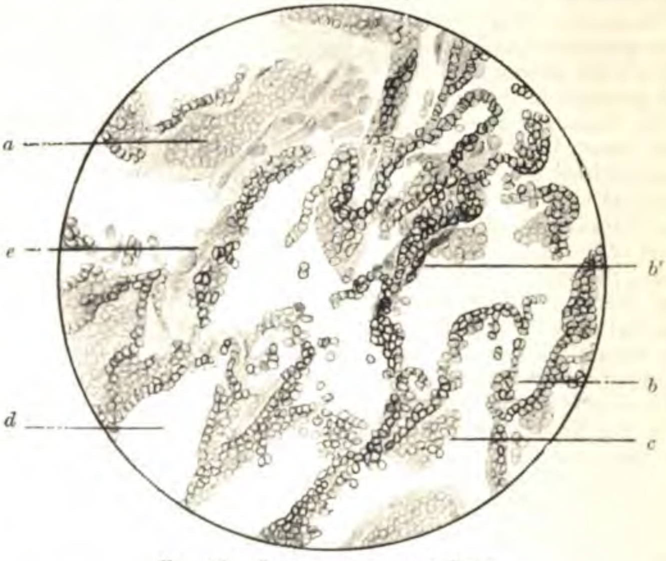


Fig. 80.—Congestion of the Lung

Showing, *a*, blood-vessel distended with red corpuscles; *b*, loop of capillary vessel projecting into alveolus; *b′*, alveolar wall obscured by distended capillaries; *c*, red corpuscles free in alveolus; *d*, empty alveolus; *e*, proliferating epithelial cells of alveolar wall.

It may also follow the sudden removal of pressure when a large trochar and a completely exhausted cylinder or bottle are used in paracentesis for pleural effusion. Congestion and œdema of the uncompressed lung may also occur in pleurisy with effusion. The condition immediately preceding the escape of blood from the vessels during the formation of a hæmorrhagic infarction is probably one of extreme congestion, and embolism of a branch of the pulmonary artery or of capillaries within the lung is probably attended by congestion of neighbouring vessels.

Chronic congestion is observed in its most typical form in long-standing obstructive disease of the mitral valve with failure of compensation.

Œdema of the lung is a further stage of the state of congestion. It is constantly observed in the post-mortem room when death has occurred from pneumonia, bronchitis, emphysema, nephritis, chronic valvular disease of the heart, or from cardiac failure in acute and chronic disease. In cases of general dropsy, if the pleural surfaces are adherent on one side of the chest, there is often œdema of the corresponding lung, whilst on the opposite side there is hydro-thorax.

Hypostatic pneumonia is a term used to describe a condition often observed at the bases of the lungs when death has resulted from exhaustion and cardiac failure. It is due to congestion, œdema, and hypostasis, in association with alveolar collapse and occasionally with the lesions of broncho-pneumonia. The term 'splenisation' is sometimes used to describe this condition.

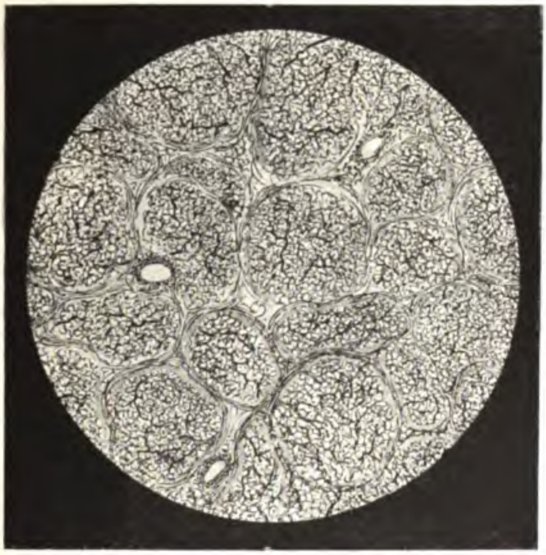


FIG. 81.—ŒDEMA OF THE LUNG

The alveoli are filled with a serous exudation, but no cells are present.

Morbid anatomy.—In acute congestion the lung on section is of a red colour, and contains an excess of blood. The condition may be seen after death from pneumonia, when the disease has involved parts which are crepitant and not yet consolidated.

The changes present in congestion of the lung are illustrated in the accompanying drawing (fig. 80.)

In chronic congestion the lung is of a brownish colo and contains much less than the normal quantity of air. The effect upon the lungs is to produce the condition known as 'brown induration,' for a description of which the reader is referred to the chapter following.

In true œdema of the lung there is a serous exudation within the alveoli (*vide* fig. 81) and in the interstitial tissues, but cellular exudation is absent. On section, a pale, slightly frothy fluid pours out, and on pressure far more may be obtained. The fluid may be blood-stained where much congestion is present. In extreme degrees of this condition the lung is quite airless, and presents a bright, glistening, gelatinous appearance. It is heavy, pits on pressure, and may readily break down. As above stated, collapse and pneumonic changes are often associated with extreme œdema.

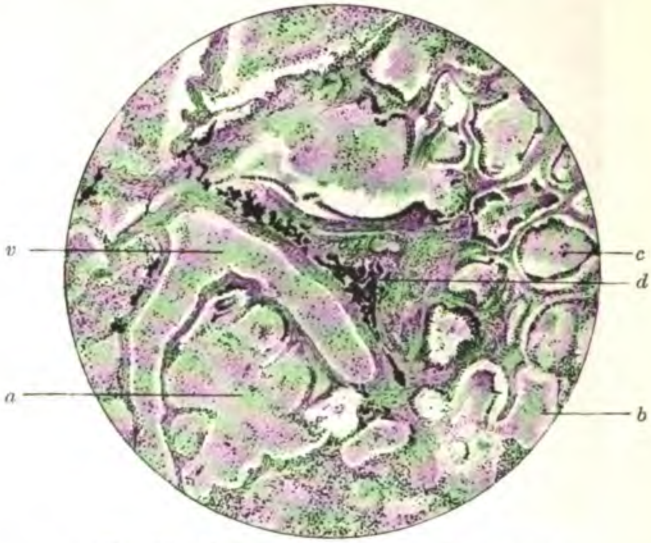


FIG. 82.—HYPOSTATIC PNEUMONIA. LOW POWER

Showing, *v*, branched vessel containing red cells and an excessive number of leucocytes; *a* and *b*, alveoli filled with œdema fluid; *c*, alveolus showing proliferation of the lining epithelium; *d*, pigment in neighbourhood of vessel.

Symptoms.—Defective aeration of the blood is a necessary result of congestion of the lungs, and dyspnœa is the chief evidence of this. Cough is almost invariably present, and is accompanied by the expectoration of mucus, which may be blood-stained, or there may be considerable hæmoptysis.

Œdema gives rise to a profuse, thin, watery expectoration, which is frothy from admixture with air. If over-distension of the right side of the heart follows, the face becomes cyanosed, and at a later period livid, the vessels of the neck are distended, and the dyspnœa is extreme. The lungs are in a condition of extreme temporary over-distension, the breath sounds are harsh, and the exudation into the bronchi produces sibilant and sonorous rhonchi. The adventitious sounds most typical of œdema are fine crackling râles at the bases of the lungs, sometimes so fine as to resemble crepitation. As the condition advances and the lower lobes become almost airless, the percussion note over the affected area becomes dull.

Cases are occasionally observed in which œdema of the lungs supervenes with remarkable rapidity during an acute illness of sudden onset. One of the kind observed by the writer in a man æt. 33 commenced with fever and pain in the chest, followed after three days by the expectoration of enormous quantities of frothy fluid. This continued until death, which occurred in less than eighteen hours from the onset of this symptom. The trachea and bronchi were filled with frothy fluid, and both lungs were extremely œdematous. Careful investigation failed to afford any adequate explanation of the exact nature of the case, and it was classified in the Post-Mortem Reports under the head of 'Acute Pulmonary Œdema.' Cases of a somewhat similar character, in which recovery has occurred after a febrile illness of three or four days' duration, have been regarded as examples of abortive pneumonia. Cases of acute œdema with a non-febrile onset, accompanied by rusty expectoration of a watery character, have been called by Traube *Pneumonia Serosa*.

Diagnosis.—The conditions under consideration are so rarely primary that their recognition is, in the majority of cases, due first to a knowledge that they are likely to occur under given circumstances, and, secondly, to the discovery of certain physical signs.

As already stated, the most typical sign of pulmonary œdema is the presence of crepitation or fine crepitant râles, especially at the bases of the lungs. Acute congestion and œdema is distinguished from embolism of the pulmonary artery by the presence of rapid panting breathing, which is in marked contrast to the prolonged deep inspiration and expiration which are characteristic of the latter condition after the disappearance of the extremely urgent symptoms which often mark its onset. From spasmodic asthma it may be differentiated by the absence of the extreme prolongation of expiration.

Urgent dyspnœa with diffuse rhonchus and sibilus in cases of mitral stenosis may be due to pulmonary congestion and œdema, and in the absence of the typical murmur a diagnosis of acute bronchitis may suggest itself if the previous history of the patient is unknown.

Treatment.—Pulmonary engorgement with cyanosis or lividity, and over-distension of the right side of the heart, from whatever cause arising, may, if unrelieved quickly, terminate in cardiac failure. Under such circumstances venesection is the obvious indication, and the abstraction of from six to ten ounces of blood often affords great relief. If the condition should subsequently recur, it may be necessary to repeat the operation. Dry cupping is also of service. Turpentine stupes should be applied to the front and back of the chest alternately, and aromatic spirits of ammonia with ether and digitalis should be given at intervals of three hours. Free purgation by calomel and salines also indirectly relieves the distension of the systemic venous system.

In conditions which are not of such immediate urgency the indications are to promote expectoration and maintain the force of the circulation, and to relieve the venous system by purgatives and diuretics.

For the treatment of the various inflammatory affections of the lungs which are accompanied by congestion reference may be made to the chapters which deal with them.

J. K. F.

CHAPTER XXV

BROWN INDURATION OF THE LUNG

(CHRONIC CONGESTION)

PAGE
Etiology 283
Morbid anatomy 283
Symptoms 284
Treatment 284

Etiology.—This condition is a result of long-continued obstruction to the flow of blood from the pulmonary veins to the left cavities of the heart, and is observed in its most typical form in stenosis of the mitral orifice. Careful observations of cases of obstruction of the mitral orifice and of incompetence of the mitral valve show that the effects of the two conditions upon the lungs and circulation are not precisely similar.

In stenosis the obstruction is, so to speak, passive, and its results are chiefly observed in the lungs; whereas in regurgitation a more decided effect is produced upon the systemic venous circulation. The clinical picture of cardiac failure in the two conditions illustrates this difference: in mitral regurgitation it is that of general dropsy, whereas in stenosis it is far more often that of extreme pulmonary congestion.

Morbid anatomy.—The lungs are generally diminished in size, unless the condition is associated with emphysema. They are firmer and heavier than normal, contain but little air, and do not collapse when the thorax is opened. Pleural adhesions are often present, especially over the left lower lobe.

The pleura is generally somewhat thickened and its surface is pigmented, but the characteristic rusty red tint is more obvious on section of the lung. Brown serous fluid may then exude, but in the most advanced stages this is not observed; in such the lung is almost dry, and similar fluid only exudes upon pressure. The exudation acquires its colour from the presence of granules of hæmatoidin of a golden brown tint, and of degenerated red-coloured corpuscles.

The section shows that the change is not uniformly distributed;

ill-defined areas of dense, drier, and more pigmented tissue are seen to merge into the surrounding less deeply brown-coloured lung. The change is usually most marked in the lower lobes. Wedge-shaped hæmorrhagic infarctions in various stages are frequently present, but do not form an essential feature of the condition.

On *microscopical examination* the alveolar walls are seen to be thickened and pigmented. The capillaries of the interalveolar septa are distended, varicose, and engorged with blood; they project into the alveoli in the form of loops, and their walls also show thickening. Within the air-vesicles are numerous epithelial cells, chiefly lying free, but some few are still attached to the walls; the cells contain reddish-brown pigment in the form of granules. Some red corpuscles are commonly present in the intra-alveolar exudation. The lymph spaces of the alveolar walls also contain black and brown pigment.

The interlobular, peribronchial, and perivascular connective tissue is thickened, and contains pigment granules, which are either free or enclosed within cells.

The mucous membrane of the bronchi, especially that of the smaller tubes, is intensely congested, and its vessels are dilated, tortuous, and filled with blood. The epithelial lining has in great part disappeared, whilst the basement membrane is œdematous and thrown into folds by the distended capillaries lying beneath it.

The vessels of the pleura are enormously distended, and in places extravasation of blood may have occurred. Patches of atheroma may be found in the larger branches of the pulmonary artery, and to a less extent in those of the pulmonary veins.

Symptoms.—It is hardly possible to distinguish the symptoms directly due to brown induration of the lungs from those attributed to the associated condition of the heart, and the attempt is seldom made. When in the later stages of a case of mitral stenosis considerable hæmoptysis occurs, it is generally due to the formation of an hæmorrhagic infarct in the lungs, and infarction and brown induration are very commonly, though not invariably, associated.

Dyspnœa and blood-stained expectoration in mitral disease may arise from temporary cardiac failure, and it is not safe to infer from their presence that the lungs are in the condition of induration.

The physical signs in the earlier stages are those described in the preceding chapter; later on there may be dulness on percussion at the bases, and the breath sounds may be weak but bronchial in quality.

Treatment.—In the cases in which this condition is present the treatment is invariably directed more to the state of the heart than to that of the lungs. The former may be directly remediable, whereas the latter can only be influenced by bringing about an improvement in the circulation of the blood.

J. K. F.

CHAPTER XXVI

COLLAPSE OF THE LUNGS

PAGE

Atelectasis 285

Mechanism of collapse 286

Causes of collapse 288

1. Obstruction of the upper air-passages 288
2. „ „ bronchi 289
3. Impaired expansion of the chest 289
4. Compression of the lung 290
5. Pneumothorax 290

Morbid anatomy 290

Changes in collapsed lung 291

Symptoms and physical signs 291

Diagnosis 292

Prognosis 292

Treatment 293

Atelectasis.—The term Atelectasis, which is not now in common use, was originally applied to the condition of partial or complete absence of expansion of the lungs after birth—*i.e.* the retention of the fœtal state—whilst Collapse was reserved for the airless condition of lungs previously expanded.

Atelectasis may be produced by a variety of conditions, such as congenital weakness or disease, bronchial obstruction by mucus or meconium, the pressure of enlarged bronchial glands, enlargement of the abdomen interfering with the action of the diaphragm, the use of forceps, and other causes. The unexpanded lung is quite healthy, and the condition cannot therefore be regarded as a pulmonary disease.

If the child has not breathed at all, the lungs present throughout the colour and texture of the adult liver.

If death has occurred after a few respirations, the surface is studded with distended air-vesicles of a bright vermilion or rose tint, and the remainder of the lungs presents the appearances of atelectasis.

It is probable that in weakly children portions of the lungs commonly remain unexpanded during the first year of life.

ill-defined areas of dense, drier, and more pigmented tissue are seen to merge into the surrounding less deeply brown-coloured lung. The change is usually most marked in the lower lobes. Wedge-shaped hæmorrhagic infarctions in various stages are frequently present, but do not form an essential feature of the condition.

On *microscopical examination* the alveolar walls are seen to be thickened and pigmented. The capillaries of the interalveolar septa are distended, varicose, and engorged with blood; they project into the alveoli in the form of loops, and their walls also show thickening. Within the air-vesicles are numerous epithelial cells, chiefly lying free, but some few are still attached to the walls; the cells contain reddish-brown pigment in the form of granules. Some red corpuscles are commonly present in the intra-alveolar exudation. The lymph spaces of the alveolar walls also contain black and brown pigment.

The interlobular, peribronchial, and perivascular connective tissue is thickened, and contains pigment granules, which are either free or enclosed within cells.

The mucous membrane of the bronchi, especially that of the smaller tubes, is intensely congested, and its vessels are dilated, tortuous, and filled with blood. The epithelial lining has in great part disappeared, whilst the basement membrane is œdematous and thrown into folds by the distended capillaries lying beneath it.

The vessels of the pleura are enormously distended, and in places extravasation of blood may have occurred. Patches of atheroma may be found in the larger branches of the pulmonary artery, and to a less extent in those of the pulmonary veins.

Symptoms.—It is hardly possible to distinguish the symptoms directly due to brown induration of the lungs from those attributed to the associated condition of the heart, and the attempt is seldom made. When in the later stages of a case of mitral stenosis considerable hæmoptysis occurs, it is generally due to the formation of an hæmorrhagic infarct in the lungs, and infarction and brown induration are very commonly, though not invariably, associated.

Dyspnœa and blood-stained expectoration in mitral disease may arise from temporary cardiac failure, and it is not safe to infer from their presence that the lungs are in the condition of induration.

The physical signs in the earlier stages are those described in the preceding chapter; later on there may be dulness on percussion at the bases, and the breath sounds may be weak but bronchial in quality.

Treatment.—In the cases in which this condition is present the treatment is invariably directed more to the state of the heart than to that of the lungs. The former may be directly remediable, whereas the latter can only be influenced by bringing about an improvement in the circulation of the blood.

J. K. F.

London in 1876[1] entitled 'A Case of Diphtheritic Paralysis simulating extensive Lung Disease.' In this paper Pearson-Irvine clearly indicated his belief that paralysis of the inspiratory muscles may lead to collapse of the lung. The following abstract of the notes of this case is quoted from Dr. W. Pasteur's paper,[2] to which reference is made in the chapter on Paralysis of the Diaphragm (*vide* p. 667).

'Y. W., a girl, aged six years, had suffered from symptoms of bronchitis for fourteen days. A brother, aged four years, had died six weeks previously of scarlet fever, and on close inquiry it was subsequently ascertained that both he and the patient herself had had diphtheria at that time. Her illness began rather suddenly with short frequent cough and considerable dyspnœa. She was taken off her feet almost at once. She was feverish the first two or three days, but had suffered no pain.

'When first seen she was dull and listless of aspect, with a queer look about the eyes, and a drooping head. The temperature was normal, the respirations (14) shallow, slow, but not laborious. She had a continuous cough, as though to clear the throat. There was decided flattening of both infraclavicular regions, with almost complete absence of movement. The flattening was extensive and uniform, and it appeared as though the upper part of the chest had fallen in. The bases expanded unusually fully, and there was a *strong action of the diaphragm*. There was marked loss of resonance above and under both clavicles as low as the third ribs, and on each side toward the sternum it terminated at the level of the second ribs in absolute dulness, in what appeared to be an increased area of superficial cardiac dulness. In the supraspinous fossa there was what appeared to be absolute dulness, but the rest of the back was resonant, the bases, indeed, being abnormally so. Every variety of bronchitic râle could be heard. At the apices there were loud sonorous râles not concealing what seemed to be true tubular breathing. There was clear whispering pectoriloquy at both apices.

'Four days later she was again brought to the hospital, having been worse in the interval. The chest signs were unchanged, the temperature remained normal. Pulse 60, respirations 14. No distress. The head drooped more, and there was decided left strabismus. The paralysis of the trunk and limbs was also more marked. The speech had become thick and nasal, and on swallowing there was distinct pharyngeal gurgling. The flattening was pointed out to the mother, who at once remarked that the child had been remarkably full chested before the present illness.

'She was in much the same state when next seen three days later. A week after this there was a striking change. The head was all but erect, the squint had nearly disappeared, the walk was no longer ataxic, and the child was quite cheerful. The improvement occurred quite suddenly. There was now an increase of resonance below the clavicles, and a distinct diminution of dulness around the cardiac area. The breathing was tubular at the right apex, weak elsewhere, with a complete absence of râles. The upper regions of the thorax still moved imperfectly. A fortnight later she was to all appearance quite cured. There still remained some impairment of movement below the clavicles, but these regions had now become more prominent. The lung gradually invaded the area of increased cardiac dulness, which became reduced to normal dimensions. Tubular breathing below the right clavicle persisted for some time longer.'

---

[1] *Clin. Soc. Trans.* vol. ix.

[2] *Internat. Jour. Med. Sciences*, Sept. 1890.

Commenting on this case Pearson-Irvine makes the following remarks:

'The disease was peculiar in many respects, particularly as regards the bronchial attack, which seemed to be so extensive as judged by physical signs, and yet so unlike in its general symptoms and course to that of an ordinary general catarrh of the tubes. . . . *All the physical phenomena met with at the upper parts of the thorax appear to me to have been due to a paralysis of the muscles concerned in the elevation and expansion of these parts.*

'In consequence of this paralysis and the increased action of the diaphragm (?) not only were the apices reduced to the expiratory condition, but even to a condition of temporary collapse, or one allowing accumulation of secretion, so that there were flattening and loss of resonance with tracheal breathing, and also retraction of the edges of the lungs, with consequent increase of the cardiac dulness.'

Fagge [1] arrived independently at the conclusion that failure of inspiratory power may produce collapse independently of bronchial obstruction. 'Whenever even a small part of the organ fails to be acted on by the forces which are concerned in inspiration, its elasticity brings about a total collapse of its substance, notwithstanding that the tubes which serve it may be patent. This, it must be admitted, is a hard doctrine to accept, but there seems to be no doubt about its truth.'

Collapse in cases of pleural effusion, apart from the presence of positive intrathoracic pressure, is to be similarly explained.

Etiology.—Collapse may occur from the following causes:

(*a*) Obstruction of the upper air-passages.
(*b*) Obstruction of the bronchi.
(*c*) Impaired expansion of the chest.
(*d*) Compression of the lung.
(*e*) Pneumothorax.

(*a*) *Obstruction of the upper air-passages.*—Chronic nasal catarrh, 'snuffles,' enlargement of the tonsils, and adenoid growths in the naso-pharynx impede the entrance of air to the glottis, and favour the production of collapse by limiting the expansion of the chest. It is a familiar observation that in children the operation for the removal of adenoid growths is followed by a more complete expansion of the lungs.

Laryngeal stenosis from any cause—*e.g.* diphtheria, laryngitis, papillomata on the vocal cords—is liable, if sufficiently long continued, to give rise to collapse of the lungs. A narrowing of the trachea from internal stricture or from external pressure may lead to a similar result; but, as such conditions are rare in childhood—the period of life during which collapse is most easily produced—they are of minor importance.

When from any of the above causes the entrance of air is impeded, the lower intercostal spaces and lower parts of the chest sink in during inspiration, and collapse results from the absence of expansion of the subjacent lung.

(*b*) *Obstruction of the bronchi.*—The causes of bronchial obstruction are various, and most of them have already been described in the chapter on Bronchial Stenosis (*vide* p. 150). Those most effective in the production of collapse, especially in children, are bronchitis, broncho-pneumonia, an extension of laryngeal diphheria to the bronchi, œdema of the lung, and whooping cough in association with laryngeal obstruction and bronchitis. The condition is also often observed in bronchitis in old people, and in cases of typhoid fever. Inflammatory swelling of the lining membrane of the bronchi, and the accumulation of mucus and pus in the tubes are the changes most commonly present.

Collapse in cases of thoracic aneurism, or mediastinal tumour, is usually due to obstruction of the bronchi.

In bronchiectasis collapse of the lobules surrounding the dilated tubes is a frequent result of obstruction caused by the accumulation of secretion.

(*c*) *Impaired expansion of the chest.*—The effect of paralysis of the muscles, or rather of certain groups of muscles concerned in respiration, has already been described in considering the mechanism of collapse. The chest walls in weak and especially in rickety children lack sufficient rigidity to allow of the normal action of the intercostal and other muscles, and to withstand the atmospheric pressure.

When in such cases the cavity of the thorax should be enlarged by the descent of the diaphragm, the intercostal spaces and lower parts of the chest recede during inspiration, and the underlying parts of the lungs tend to collapse. This is naturally more noticeable in the presence of any affection, such as bronchial catarrh, tending to obstruct the entrance of air into the lungs. A sufficiently long continuance of the condition leads to permanent alteration in the shape of the chest, either in the form of the 'transversely constricted' thorax, the pigeon breast, or the rickety chest. The former is characterised by a depression on both sides which starts from near the xiphoid cartilage and passes outwards and slightly downwards to the mid-axillary region.

In conditions of extreme weakness, whether from old age or prolonged fever, or other cause, collapse of the bases of the lungs is of frequent occurrence, and in such cases is chiefly due to impaired expansion of the chest.

(*d*) *Compression of the lung.*—Compression of the lung by fluid in the pleural cavity is a very common cause of collapse, and the condition may also arise from the pressure of an aneurism or a solid growth originating either in the mediastinum or in the lung. The distension of the abdomen from any cause may produce a similar result, either from direct pressure or by opposing the descent of the diaphragm and thus preventing the due expansion of the lower lobes.

(*e*) *Pneumothorax.*—Partial collapse of the lung is the immediate effect of making an opening into the pleura. This is due partly to the elasticity of the organ, and in part to the fact that it is no longer acted upon by the forces which produce expansion of the chest. Complete collapse may follow the accumulation of air in the pleural cavity, particularly if a condition of positive intrapleural pressure is established. Subsequent acute inflammation of the pleura leading to the formation of a pyopneumothorax will further aid in rendering the lung airless.

Morbid anatomy.—The appearances presented by the lobular form of collapse are very characteristic. If situated upon the surface, the area is sharply defined, angular in outline, shrunken, depressed, and of a dark violet tint, and on section it has a non-crepitant, smooth, and glistening surface. On pressure a small quantity of serum may exude, but the tissue does not break down. It is commonly somewhat congested. An area of collapse, if not of long standing, may be restored to a crepitant condition by insufflation; it then becomes of a bright red tint.

Considerable tracts may be thus affected, or numerous small patches may be seen upon the surface of the lung, or at the margins of the lower lobes, the most common sites of the change. The parts of the lungs lying beside the bodies of the vertebræ often present extensive areas of collapse, which may be symmetrical and assume a pyramidal form.

Sometimes, and particularly in children, almost the whole of a lobe—especially the middle lobe of the right lung—may be found in a condition of collapse. This may have originated from the confluence of numerous separate areas, as bronchial obstruction became more and more extensive. Collapse is more marked, as a rule, upon the surface of the lung than in the deeper parts. The bronchi leading to areas of lobular collapse are usually obstructed by purulent secretion. Other appearances will vary with the nature of the case.

The lung tissue around areas of collapse is very often œdematous. It may, however, have undergone emphysematous enlargement. The collapse of the alveoli renders the bronchi more obvious on section, and they project from the surface.

A lung completely collapsed and compressed by pleural effusion is quite airless. It sinks in water, is of a pale slate-blue colour, very tough, and not so distinctly glistening as in the lobular form.

Changes in collapsed lung.—After recovery from an acute illness which has been accompanied by pulmonary collapse—*e.g.* an attack of bronchitis—as respiratory power is regained, air may again enter the affected parts of the lung, and they may be restored to the normal condition.

If, however, the change is permanent, the alveoli remain compressed and obliterated, and a chronic inflammatory process affecting chiefly the connective tissue structures is apt to follow. A small-celled exudation is found around the bronchioles, and the interlobular and interalveolar tissues become thickened. Subsequently the smaller bronchi may undergo dilatation, and be seen on section to be filled with yellow pus.

In other cases the part undergoes an atrophic change, and later on a puckered scar may indicate its site.

Extensive atelectasis in the newly-born, by obstructing the pulmonary circulation, may prevent the closure of the ductus arteriosus and the foramen ovale, and may induce cyanosis, with dilatation and hypertrophy of the right ventricle, and thickening of the pulmonary artery. Clubbing of the fingers and toes, and signs of venous congestion in the lungs and the kidneys may also be observed in such cases.

Symptoms and physical signs.—Complete absence of expansion of the lungs in a newly-born child is obviously incompatible with life. A degree of atelectasis short of this is characterised by feebleness, arrest of growth, and symptoms indicating defective oxygenation of the blood, such as dyspnœa with rapid, shallow respiration, and a varying amount of cyanosis, with drowsiness, twitchings, and later on convulsions.

The temperature is low, the cry feeble, and general weakness is marked. The intercostal spaces and the lateral and inframammary regions of the chest recede during inspiration, and, if the child lives sufficiently long, the thorax soon becomes deformed, the sternum prominent, and the vertical and oblique sulci well marked.

Viewed from behind, the lower ribs are seen to be drawn towards the spine with each inspiration. The percussion-note over the bases may be wanting in resonance, and on auscultation the breath sounds are found to be weak, and some fine crackling râles may be present.

The probable duration of life of such children may be measured by the degree of cyanosis present; if considerable, it is rarely more than a few days, but in less severe cases death may not occur for a fortnight, or even for some months.

When collapse of the lungs occurs as a complication of bronchitis, or broncho-pneumonia, or other affections accompanied by obstruction of the smaller bronchi, its onset may be almost sudden, and marked by increasing dyspnœa with very rapid breathing and a quick and feeble pulse. The cough becomes weak and ineffective, and gradually cyanosis and other signs of defective aeration of the blood appear; the patient sinks down in the bed, and consciousness is lost.

In such cases its presence is more often inferred from the increase in the severity of the symptoms, and the inspiratory recession of the interspaces and lower parts of the chest, than from the discovery of very definite physical signs, as, owing to its lobular and scattered distribution, the percussion-note may not be much altered, particularly if emphysematous changes occur in the neighbouring lobules.

In children it is particularly important to auscultate the lower part of the chest during the deep inspiration which follows a cry or a paroxysm of coughing, as the absence of the breath sounds is then more readily appreciated.

No adventitious sounds are produced in an area which is completely collapsed, but it is often possible to recognise the presence of partial collapse from the occurrence of very fine râles at the end of a deep inspiration, caused by the entrance of air into vesicles not used in quiet breathing.

Should, however, a considerable area of the lower lobe become collapsed, dulness may be found on percussion, probably close to the spine and extending upwards, with an absence of the breath sounds. If recovery follows and the collapsed areas again expand, the chest, although much distorted, may in time almost regain its normal outline.

When extensive collapse follows the obstruction of a single large bronchus, the inspiratory recession and other signs are limited to the side affected.

Diagnosis.—When the greater part or the whole of one lung is in a condition of collapse from pleural effusion, the presence of distant tubular breathing over the affected side may lead to the diagnosis of pneumonia, a mistake which is more likely to occur in the case of a child than an adult. As this point is fully discussed in the chapter on Pneumonia (*vide* p. 226), it is sufficient to mention it here.

Collapse of the left lower lobe in valvular disease with great cardiac hypertrophy, or in pericarditis with large effusion, has not infrequently been mistaken for pneumonia. In this, as in the preceding condition, the patency of the large bronchi, owing to the resistance of their cartilages to pressure, may render the breath sounds bronchial over the area of collapse. To know of this source of error is the first step towards avoiding it.

If such signs are discovered, careful inquiry should be made into the history of the case. The febrile symptoms marking the onset of pneumonia will be absent, the sputum will not be rusty, there will probably be retraction of the affected side, and the breath sounds, although bronchial as a rule, lack the high-pitched whiffing quality combined with apparent nearness to the ear which is characteristic of pneumonia.

Prognosis.—In atelectasis, the degree of cyanosis present is, as already stated, the most sure indication of the gravity of the case. If this is gradually increasing, a fatal termination is almost certain; its progressive diminution, on the other hand, is of favourable import. In collapse occurring as a complication of the acute diseases mentioned, the prognosis is always grave, and particularly so if the child is the subject of rickets.

The chief factors to be considered are the strength of the patient, the amount of inspiratory recession, and the degree of dyspnœa and cyanosis present.

If recovery follows, the child may be left with pulmonary changes favouring the subsequent development of bronchiectasis or tuberculosis, and unless the lung undergoes re-expansion, the deformity of the chest present during the acute attack will become permanent.

Treatment.—If respiration is not satisfactorily established in a newly-born child, deep breathing may be excited by placing it in a hot bath (100° F.), and suddenly pouring cold water on the chest, or by flicking the chest with a towel wrung out of cold water, or artificial respiration may be used. Slapping the nates and tickling the fauces are approved methods of exciting the act of breathing. Mustard and stimulating liniments applied to the chest have a similar effect. It is of course important to see that the upper air-passages are free from obstruction.

In such cases warmth is essential, but it is equally important not further to impede respiration by surrounding the chest with many yards of flannel. When the child is not in the warm bath, a layer of cotton-wool should be warmed and lightly applied to the chest.

When collapse occurs in the course of some acute disease, the treatment to be adopted will necessarily depend very much upon the nature of the primary affection; but the general indications are the same in all cases, viz. to promote expectoration and to maintain the strength of the patient. These ends are best fulfilled by rest in bed in a warm and slightly moist air, and the administration of carbonate of ammonia, squills, stimulants and liquid food. Dr. Pasteur has drawn attention to the great value of artificial respiration in collapse from paralysis of the diaphragm, and the method is equally applicable when it depends upon other conditions. An emetic of mustard and water or ipecacuanha (5 grs. of the powdered root or ʒj of the vinum) may be given to a child, as during the act of vomiting much mucus may be got rid of. It is, however, unnecessary to repeat what has already been stated in the chapter on Bronchitis on the subject of the administration of emetics in such cases.

All debilitating conditions in childhood necessarily predispose to collapse during acute pulmonary affections, and are therefore as far as possible to be avoided. For the same reason, bronchial catarrh in the subjects of rickets should always be regarded seriously, no matter how slight it may at first appear.

In cases of collapse secondary to pleurisy, much benefit may be obtained from the use of gymnastic exercises, which tend to expand the chest and develop the thoracic muscles. A residence at a high altitude, by necessitating deeper breathing, also helps to open up unused portions of the lungs. The inspiration of air from Waldenburg's pneumatic apparatus promotes expansion of the lungs, and is well worthy of trial.

The general plan of treatment of all cases after recovery from acute illness attended by collapse of the lungs should be of a tonic character.

J. K. F.

CHAPTER XXVII

PULMONARY TUBERCULOSIS

PAGE
Pathology of tubercle 296
Morbid anatomy of tubercle 299
    Structure 299
    Formation 300
Subsequent changes in tubercle 302
    Caseation 302
    Fibrosis 304
Etiology of pulmonary tuberculosis 304
    Infection 304
    Direct transmission 309
    Predisposing conditions 311
    Influence of heredity 311
    Loss of resisting power 313
    Race and climate 314
    Age and sex 314
    Previous disease 315

Definition.—A disease of the lungs due to the presence of a specific organism, the bacillus tuberculosis.

The prolonged controversy as to the nature of tubercle may be said to have terminated with the general acceptance of the views of Koch (published in 1882).

Into the history of this controversy we do not propose to enter; the subject has been exhaustively treated by many writers, and may now be omitted from a work which is intended to have a practical character.

Concurrently with that just alluded to, and inextricably mixed up with it, another and more heated controversy has raged as to the relation of tubercle to the disease known as 'phthisis' or 'consumption.' The writer has for many years urged that these terms should no longer be used, and that when tubercle is present in the lungs the most appropriate name for the disease is 'pulmonary tuberculosis;' and he has observed of late, with some satisfaction, signs of a disposition to adopt this nomenclature.

In advocating this change he expressly refrained from affirming that all cases included under 'phthisis pulmonalis' are tuberculous, although such is undoubtedly true of the great majority of them;

the contention is that, when tuberculous disease is present in the lungs, the name given to the affection should be one as to the significance of which there can be no dispute.

To some the question of a name may appear a matter of very small importance. Phthisis is no doubt a 'good old-fashioned term,' as it has been described by one who did not wish to part with it, but the word is surrounded by such a haze of doubt that so long as it remains in use there is little likelihood that our knowledge of the chronic inflammatory and destructive diseases of the lungs, not of tuberculous origin, will make any real progress. Many advantages attend this change of nomenclature. The use of the term 'pulmonary tuberculosis' harmonises the terminology of all tuberculous affections; it obviates the constantly recurring necessity for defining one's attitude on the question of the relation of tubercle to 'phthisis;' it gets rid of that most misleading phrase 'the stages of phthisis;' it necessitates the use of all known methods for determining the presence of tubercular disease before coming to that diagnosis; and it induces a closer study of all cases of destructive disease of the lungs which are proved by repeated examination not to be tubercular. Such study must have shown to others as it has to the writer that cases of the kind are more numerous than is supposed, and that much remains to be done before their true nature is understood.

PATHOLOGY OF TUBERCLE

The proofs given by Koch in 1882 that the bacillus tuberculosis is the essential cause of tubercle were singularly complete, and he left little to be done by subsequent workers, except to repeat his experiments and confirm his results.

The **bacillus tuberculosis** is a thin rod-shaped organism, measuring from 2 to 4 micro-millimetres in length and about 0·20 μ in breadth. The rods may be straight, but are usually slightly curved or bent and present rounded extremities. The bacilli as a rule occur singly, but two may be united, either at an obtuse angle or so as to form a straight line. A long row may be thus formed in a cultivation, but is rarely found in the tissues. In preparations containing tubercle bacilli stained by any of the methods subsequently to be described, the bacilli appear to be composed of round or rod-shaped granules of deeply stained protoplasm contained in a sheath which is less deeply coloured. Clear globular unstained spaces occur at regular intervals, which give the organism a dotted or beaded appearance: from three to six or eight of these may be present in a single bacillus. This appearance, which is most commonly seen in tubercular sputum, but is also found occasionally in the bacilli in sections and in old cultivations, was at one time thought to be due to the presence of spores at the unstained points; but this view is no longer held, and the absence of coloration is attributed to the fact that at those points the sheath is empty. The presence of spores in tubercle bacilli is, however, considered as certain by many observers, but the spores have not so far been identified.

Conditions of growth.—The range of temperature suited to the growth of the bacillus is between 30° and 40° C., the most active changes taking place at the temperature of the blood (37° C.).

If sterilised blood serum is inoculated with tubercular material and kept at the required temperature, after an interval of from ten to fourteen days a growth appears on the surface of the medium; this is at first a thin white deposit which gradually increases in size, becomes of a slightly yellow tint, and forms at a later period a dry scale-like film. The organism is aerobic—that is, it requires free oxygen for its growth—and it does not liquefy the medium. The growth is slow and lasts for a short time only, a week or more. Young colonies on solid media show at the margin wavy hairlike bundles, and in the centre twisted bundles composed of bacilli lying parallel to one another.

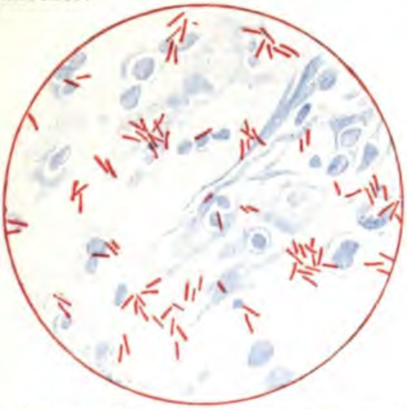


FIG. 83.—TUBERCLE BACILLI IN SPUTUM

On glycerin-agar growth is more rapid and colonies appear at an earlier date. Growth also takes place on the surface of a potato if it is moistened and kept at a suitable temperature. An indefinite series of cultures may thus be made and the disease reproduced from them in animals.

Although tubercle bacilli require a high temperature for their growth, they retain their vitality for long periods at ordinary temperatures. Sputum, whether moist or dry, has been proved to contain living bacilli two months after its discharge.

Putrefaction has no effect upon their virulence, but they are killed by moist heat at a temperature of 100° C., or by carbolic acid (2 to 3 per cent.) in a few minutes.

Bacilli in dried sputum are killed if exposed to dry heat for an hour at a temperature of 100° C.

The bacilli are present in all recent and rapidly growing tuberculous lesions, the number as a rule varying directly with the activity of the process. In such as are obsolete or have undergone caseation or fibrosis it may be impossible to demonstrate the presence of the virus in any other manner than by inoculation, and in some fibrous lesions undoubtedly tubercular even this test may fail.

Septic organisms are often present in association with the specific virus of the disease, and are in part responsible for the inflammatory and suppurative changes by which it is accompanied.

Staining.—Koch's original method of staining was as follows: 1 cc. of a saturated solution of methylene blue is shaken up with 200 cc. of distilled water, and to this 0·2 cc. of a 10 per cent. solution of potash is added, and the solution is again shaken. Cover-glass preparations of sputum or tissue containing the bacilli are placed in this for twenty-four hours. They are afterwards decolorised in a saturated solution of vesuvin. By this process the bacilli are stained blue and the background black.

The methods for staining tubercle bacilli are now so numerous that it is impossible to give them in detail; the principle, however, is the same in all. The organisms are first deeply coloured by a basic aniline dye, the action of which is aided by a mordant, a substance which has the property of fixing the dye more firmly upon the bacillus. A reagent is subsequently added which decolorises the surrounding tissues but does not affect the bacilli, which retain their colour. Various decolorising agents are in use, such as vesuvin, nitric acid, and alcohol.

At the Brompton Hospital, where a large number of examinations are made daily, the following method is adopted. A small portion of sputum which appears most likely to contain bacilli is placed upon a cover-glass, upon this another cover-glass is firmly pressed and the two are then separated. They are then passed three times through the flame of a Bunsen burner. A solution composed of water 95 cc., phenol 5 cc., saturated alcoholic solution of fuchsine 10 cc., is heated in a watch-glass on a sand bath until steam rises, and the cover-glasses are floated on the surface for about four minutes. They are then placed in a 25 per cent. solution of nitric acid, and allowed to remain in it until all colour disappears. They are then washed in tap water. The glasses are then placed for counter-staining in a saturated solution of methylene blue, washed again in tap water and allowed to dry; when dry they are mounted in Canada balsam. This method is rapid, and the results obtained are trustworthy.

Forceps should be used for removing the cover slips from the solutions.

By this method the bacilli are stained red, and pus corpuscles and other organisms which may be present blue.

The bacilli, if in large numbers, are generally in clusters, and are easily recognised with a magnifying power of from 200 to 300; when only a very few are present, a substage condenser and a $\frac{1}{12}$ oil immersion lens may be necessary, and should be used in all doubtful cases. The morning expectoration should in such cases be examined daily.

Tubercle bacilli outside the body.—It has been shown that tubercle bacilli may be present in the dust of rooms occupied by patients suffering from pulmonary tuberculosis, and that organisms so obtained may be virulent. Osler cites a case in which dust from the wall in the neighbourhood of the bed, in a room inhabited by a tuberculous patient, was infective six weeks after the death of the patient. In many cases, however, no results have followed upon the inoculation of animals with dust obtained from presumably infected places which is certainly more likely to hold the specific organism than that which may be inhaled in the public streets and other places.

Such facts appear to show that there exists very little ground for the loose statements commonly made as to the almost universal prevalence of the tubercle bacillus; such, for example, as that 'we all take the tubercle bacillus into our lungs in numbers every day.' It may be so, but there is no proof of it, and all recent research tends to an opposite conclusion. This is a point of much importance, for if the distribution of the bacillus be universal, it follows that it may be a hopeless task to attempt to diminish the prevalence of the disease by measures tending to check infection.

MORBID ANATOMY OF TUBERCLE

Structure of a tubercle.—Three varieties of cells are met with in tubercles: (*a*) epithelioid cells, (*b*) giant cells, and (*c*) leucocytes.

(*a*) Epithelioid cells are of large size, and contain a single vesicular oval nucleus with clear nuclear juice and nucleoli; the protoplasm of the cells is coarsely granular.

(*b*) Giant cells are of much larger size; they consist of coarsely granular protoplasm, and contain numerous oval nuclei which are often arranged in a row near the periphery or may be gathered together at one pole of the cell. Giant cells are irregular in shape and present radiating processes at their margins. They are often situated near the centre of a tubercle; but may be found elsewhere, and several may be found in a single tubercle.

(*c*) Leucocytes, or 'lymphoid' cells, are round cells consisting of finely granular protoplasm with a small round nucleus. These cells are in all respects similar to those ordinarily present where inflammation is in progress.

Tubercles in an early stage consist almost entirely of cells of the epithelioid type, which are now held to be the typical cells of tubercle. They may contain bacilli in their interior at the earliest stage of the process and before the emigration of the leucocytes has commenced.

At a later period the leucocytes penetrate the nodule and may then be in such numbers as to mask the presence of the epithelioid cells; this condition is known as the 'small-celled' or 'lymphoid' tubercle.

As the tubercle increases in size a fine fibrillary reticulum appears at its margin, the fibrils lying between the epithelioid cells. At a later period the outline of the tubercle becomes more sharply defined, owing partly to the cells at the periphery becoming to some extent flattened by pressure from within, and partly to the fact that towards the margin the reticulum becomes condensed.

Formation of a tubercle.—The origin of the elementary structures which enter into the formation of a tubercle, and the share taken by the connective tissue cells and the migratory cells respectively in its production, have long been and still are matters of controversy.

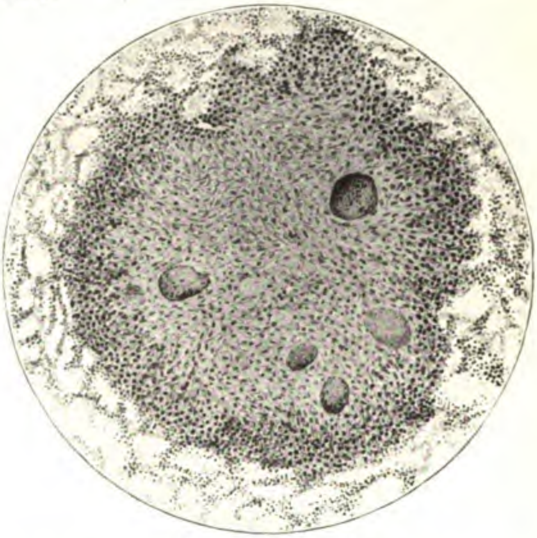


FIG. 84.—TUBERCLE

Showing leucocytes chiefly at the margin; epithelioid cells and giant cells.

According to Ziegler and Koch the cellular elements of a tubercle are chiefly derived from the white corpuscles which have left the vessels, the share taken by the fixed cells of the tissues being of secondary importance. Arnold, Baumgarten, Weigert, and others have, however, shown that the latter play a much more important, and indeed a preponderating *rôle* in its formation. This view is now accepted by most pathologists.

When tubercle bacilli obtain a lodgment in the tissues of the lung or elsewhere, they multiply and produce in the nuclei of the fixed cells of the part the changes characteristic of karyokinesis. Proliferation of the nuclei and division of the cell protoplasm lead to the formation of new cells, which gradually assume the epithelioid character. These are rounded, polygonal, or cubical in form, and present a clear oval vesicular nucleus. These changes are not limited to the connective-tissue cells, but occur also in the epithelial elements of the part and the endothelial cells of the capillaries. Some of the cells undergoing proliferation contain one or more bacilli, but the majority are free from organisms.

Subsequently leucocytes leave the neighbouring vessels and surround the epithelioid cells in gradually increasing numbers. The migratory cells of the body are of various kinds, and recent researches following those of Metschnikoff upon phagocytosis have thrown much light on their properties and functions, particularly with regard to their action upon micro-organisms and foreign bodies.

The following varieties are recognised:[1]

Lymphocytes, or immature leucocytes, which resemble the elements of lymphoid tissue, and are non-phagocytic.

Hyaline cells (macrophagocytes), containing a round or uniform nucleus and abundant non-granular protoplasm. These are actively amœbic and phagocytic, but are rare in blood (2 per cent.), though abundant in the body fluid.

Coarsely granular oxyphile cells, with horseshoe-shaped nuclei, staining deeply with acid aniline dyes. These, like the last variety, are few in number in the blood, but are abundant in the body fluid, in serous cavities and areolar tissue spaces. They possess amœboid properties, but are non-phagocytic.

Finely granular cells (microphagocytes), smaller than the last variety, polynuclear, staining with acid dyes. These are abundant in the blood, and are actively amœboid and phagocytic. This is the most common form of leucocyte and pus cell.

Coarsely and finely granular cells, staining with basic dyes. The former are non-phagocytic and absent from human blood; the latter are present in small numbers (1 to 5 per cent.).

The hyaline cell is the only form of leucocyte which is believed to undergo the proliferative nuclear changes characteristic of karyokinesis.

The mode of formation of the *giant cells* of tubercles is still a matter of controversy. According to Baumgarten and Weigert they result from the nuclear proliferation of a single epithelioid cell under the influence of an irritation which is insufficient to affect the cell protoplasm. By others the giant cells of tubercle are regarded as plasmodia formed by the fusion of many smaller cells.

Metschnikoff considers that both the epithelioid and giant cells are types of phagocytes, that is, of cells which engage in a struggle for mastery with the bacilli, and, when successful, destroy them by a process of intracellular digestion.

In giant cells, as already stated, the nuclei are found at the periphery of the cell, either occupying its whole circumference, or grouped at one of the poles, leaving a considerable area of the cell free. The bacilli are generally seen either between the nuclei and the free area, or in the intervals between the nuclei; they are only rarely present in the part of the cell which is void of nuclei.

In tubercles which are undergoing caseous degeneration a similar arrangement of bacilli and nuclei is generally found;

---

[1] *Vide* art. 'Inflammation,' by J. G. Adami. Allbutt's *System of Medicine*, vol. i. p. 79.

[illegible] of the epithelioid cells and [illegible] are present in [illegible]. [illegible] and [illegible] the periphery of a tubercle [illegible] are present [illegible]. According to [illegible] regarded as [illegible] the same [illegible] of the [illegible] of the [illegible] cells [illegible] miliary [illegible].

[illegible] the [illegible] of micro-organisms [illegible] action [illegible] attributes the [illegible] the joint action of the [illegible] and the [illegible] of the [illegible].

The [illegible] action of the serum and blood [illegible] after the blood has been withdrawn [illegible] while it is circulating within the vessels, is [illegible] either by a process of [illegible] destruction, and [illegible] protecting the body from [illegible] micro-organisms.

Subsequent changes in tubercles in the lungs.—1. It [illegible] either before or after it has [illegible]. 2. It may undergo fibroid [illegible], a process which is followed as a rule by the deposit of [illegible]. [illegible] may attack only a few granulations, or by its [illegible] a tubercular lesion.

Which of these [illegible] depends to a great extent upon the [illegible] of the individual. If the resisting power is [illegible]; if on the other hand it is considerable, the [illegible]. If the infection of the lungs [illegible] during a period of ill-health of a temporary nature, it is possible that when the normal resisting power of the body has returned, the [illegible] or groups of tubercles which have [illegible], may, if completely caseous, become encapsuled; and if only caseous in part, or still of the miliary character, they may undergo fibroid transformation.

The process of caseation.—After a certain time the cells occupying the centre of a tubercle become swollen and irregular. They lose their outline, their nuclei disappear, the cells fuse together and form a structureless mass. This process is a form of necrosis—the coagulative necrosis of Cohnheim—and constitutes a step towards caseation.

The cell elements, after the process of coagulation has occurred, become condensed and undergo fatty degeneration. All traces of the structure of the tissue are lost, and the area becomes opaque and homogeneous and of a yellow colour and firm consistence, characters which suggested its similarity to cheese. When a number of separate tubercular foci have undergone this change, a caseous area of variable extent is the result. Such a mass may then soften and be discharged through the bronchi, a cavity resulting, or it may excite inflammatory changes around, which end in the formation of a fibrous capsule.

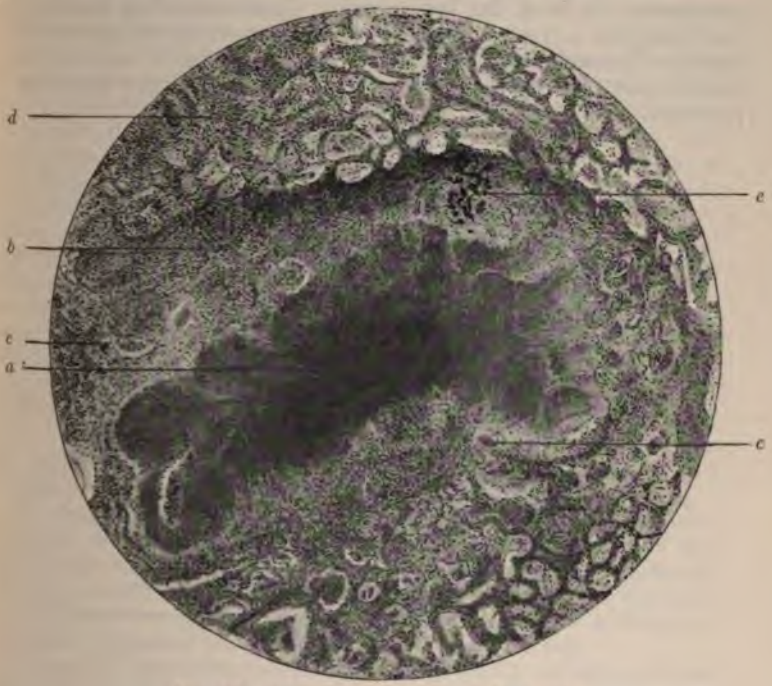


FIG. 85.—TUBERCLE UNDERGOING CASEATION

a, caseous central area ; *b*, cellular peripheral area ; *c*, *c*, giant cells ; *d*, small-celled infiltration of surrounding alveolar walls and spaces ; *e*, pigment.

In such a condition it may remain for the life of the individual, or the capsule may rupture owing to changes in its neighbourhood, and the discharge of the still virulent caseous material may set up a widespread tuberculosis of the lungs. The writer has recorded a case in which the breaking down of an encapsuled caseous mass, which had probably existed for forty years, caused acute tuberculosis of the lungs. A caseous mass corresponds to the crude or yellow tubercle of old writers.

Caseous masses frequently become hardened by the deposit of lime salts, and in such a condition they may be expectorated or found in the lungs after death.

Fibrosis.—The process of coagulative necrosis may proceed to a limited extent, but instead of the tubercle becoming caseous it may become transformed into a small, firm, fibroid nodule, still retaining its normal outline. This change is produced by an overgrowth of the connective tissue elements and the formation of fibroid tissue both within the tubercle and in the neighbouring structures. It is of far less common occurrence than caseation, and it implies a greater degree of resisting power on the part of the tissues. Tubercle bacilli may be present in small numbers in fibroid nodules; but when the lesion is very chronic they can rarely be found.

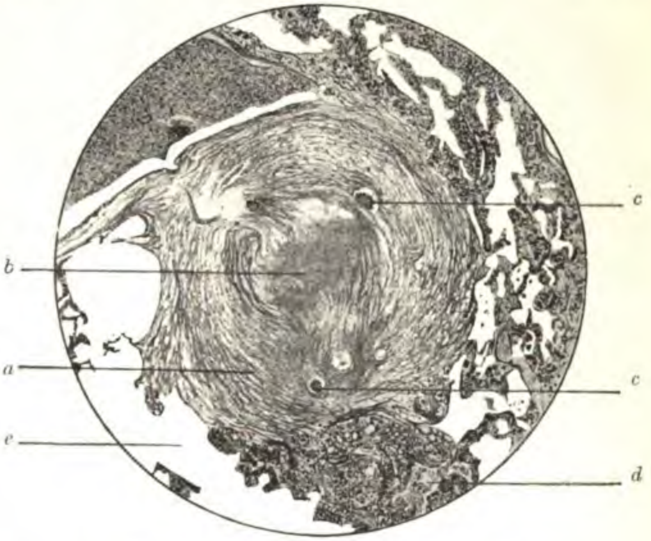


Fig. 86.—Tubercle undergoing Fibrosis

Showing, *a*, periphery, completely fibrous; *b*, central area, showing absence of complete fibrosis; *c*, *c*, remains of giant cells; *d*, fibroid induration of surrounding lung; *e*, emphysema in neighbourhood of tubercle.

ETIOLOGY OF PULMONARY TUBERCULOSIS

INFECTION

The circumstances which chiefly influence our perception of the dependence of a disease upon some specific cause are the length of the interval between the absorption of the virus and the appearance of the symptoms, the facility with which infection can be traced to its source, and the proportion of the community which is, under ordinary conditions, susceptible to the disease.

In all these respects the virus of tubercle may be said to stand at a disadvantage. The incubation period is long, and the longer the period of incubation the more difficult it becomes to trace the source of infection; whereas when it is short the contrast between the state of health and of disease is so marked as to suggest that the illness is due to infection.

The susceptible proportion of the community is, under ordinary conditions, much smaller than in the case of the specific fevers; whereas nothing demonstrates so clearly the relation of cause and effect as when a large number of individuals fall victims to a disease conveyed to them by a single infectious person.

It cannot be doubted that if tuberculosis in any way resembled the infectious fevers as regards its period of incubation and the facility with which the disease is communicated from one individual to another, its dependence upon some specific cause would have been recognised long before such was the case, although the nature of the virus might have eluded discovery. If the direct relationship between the bacillus tuberculosis and the pathological product known as tubercle be, as it now is, accepted, it follows that without infection there can be no tuberculosis, and that in every case in the human subject the disease must be either congenital or the result of infection. From this position there is no escape. We shall reserve the consideration of the subject of the direct transmission of the disease from parent to offspring until later, contenting ourselves here with the remark that, although there can now be no doubt that such an event may happen, it has not yet been shown that it is not extremely rare. When the disease is the result of infection the lungs may be involved either primarily and from without, or secondarily and from within the body.

In considering the etiology of tuberculosis, *infection* must therefore be regarded as the *causa sine qua non*, but it is not necessarily of most importance from a practical point of view. If of a large number of persons exposed to infection only a few acquire a disease, the susceptibility of the individual becomes a factor in causation of greater moment than exposure to infection.

As in the vegetable kingdom the seed and a suitable soil are necessary for the growth of a plant, so in the human body there can be, under ordinary conditions, no development of tubercle unless the specific bacillus finds a suitable environment. We repeat 'under ordinary conditions' to make it clear that, in our opinion, every individual, no matter how robust his health, is susceptible to the disease, provided the dose of the virus is sufficiently large.

The resisting power of the individual is dependent upon (*a*) his constitution, (*b*) his state of health at the time of exposure to infection, (*c*) the quantity of the virus absorbed, and possibly upon (*d*) the degree of activity of the virus.

Tuberculosis is, so far as is known, a disease of animal life, and its continued existence must therefore depend upon the transmission of the virus from one animal to another. The fact that the tubercle bacillus grows upon vegetable matter, such as potatoes, bread, and

What, again, could be more instructive on this point than the history of medical opinion as to the true nature and mode of the spread of cholera? How strongly some in authority, especially in India, have opposed the view that it spreads from man to man along the lines of human travel, and how complete has been the recent demonstration that it may be thus traced across a continent and its daily progress noted! The beneficial effects of this change of view are already apparent in the success of the measures now adopted to prevent its entrance into and spread in this country, and in its diminished prevalence in Egypt.

It is highly probable that like results will in course of time follow upon the demonstration of the dependence of tuberculosis upon a specific virus and upon the adoption of such measures for preventing its spread as are suggested by our knowledge of the way in which it may be communicated from one individual to another.

Some are deterred from accepting the view of the possibility of infection in tuberculosis from a fear that it may change the hitherto sympathetic attitude of the relatives towards the sufferer, but this is unlikely to result when the simple nature of the necessary precautions is understood; and even if the possibility is admitted, it may be pointed out that it is a recognised law of civilised life that anyone subject to a communicable disease must adopt all measures known to prevent its spread to others.

If it can be shown that by a vigorous adoption of all measures tending to limit the occurrence of infection the endemic prevalence of the disease is likely to be gradually diminished, it is clearly the duty of the medical profession to make known to the community the facts as to the mode of spread of tuberculosis, and to urge tuberculous patients to take all the necessary precautions to avoid the possibility of transmitting the disease.

Infection from food.—As we are here concerned only with tuberculosis of the lungs, the subject of infection from food is of minor importance, as probably in all such cases the lungs are affected secondarily to other organs.

Milk is one of the most active vehicles for the virus of tubercle, and there can be little doubt that the great frequency of tubercular disease of the mesenteric glands and of the intestines in children is due to infection from this source. Dr. Sidney Martin,[1] as a result of his important researches on this question, concludes that 'when tuberculosis of the cow affects only the internal organs and not the udder, the milk is not infective, but that in all cases where the udder is tuberculous the milk becomes infective, and is indeed often extremely virulent. Moreover the butter, butter-milk, and skimmed milk obtained from this class of milk is also extremely infective. There is no question therefore that this class of milk ought not to be used as food, and it is certain that its exclusion would lead to a great diminution in the large number of cases of tuberculosis that occur in children.'

---

[1] *Report of Royal Commission on Tuberculosis.*

The results obtained by inoculating and feeding with meat or meat-juice from tuberculous animals, point to the fact that the meat is infected during the dressing of the carcase, and that the virus is not present in the muscular tissue.

Inoculation.—The possibility of inoculating tubercle is now admitted, but the point is of little importance as regards pulmonary tuberculosis. Patients with pulmonary tuberculosis and tubercular ulcers of the tongue and lips may, however, transmit the virus of tubercle to others during the act of kissing.

THE DIRECT TRANSMISSION OF TUBERCLE

It has been proved as regards both man and the lower animals that tubercle may be transmitted directly from parent to offspring, and it is held by some that the virus having been thus transmitted may be dormant for a certain period and may subsequently be awakened into activity by various conditions depressing the health of the individual, and may then infect the lungs and other organs. Baumgarten, with whose name this theory of latency is now generally identified, although it was not originated by him, is prepared to go still further in this direction, as he believes that the virus may be latent for several successive generations and that a child may have been infected by a tuberculous grandmother, the mother having shown no signs of the disease.

Baumgarten's views are, briefly, that direct transmission from the parent to the offspring is by far the most common cause of tuberculosis; that the mode of transmission may be either 'germinal' or intra-uterine; that lesions are absent in the fœtus because the bacilli remain dormant for long periods in the lymphatic glands and in the medulla of bones and elsewhere; that this latency is due to a special resisting power of fœtal and young tissues, wherein cell energy is active; and that the influence of the inhalation of infected air as a cause of pulmonary tuberculosis, has been greatly exaggerated.

The evidence in favour of the direct transmission of tubercle is as follows:

1. Tubercle bacilli have been found in the calf in cases in which the cow was tuberculous (von Johne, von Malvoz, Brouvier).

2. The bacillus tuberculosis has been found in the placenta, in the fœtal placental villi, and in the fœtus removed by Cæsarean section, in cases in which the mother was tuberculous. Of such cases there are now a considerable number on record.

The following were observed by Birch-Hirschfeld, Schmorl, and Kockel:[1]

Case I.—A fœtus removed by Cæsarean section from a tuberculous mother. The placenta contained tubercle bacilli.

Case II.—A pregnant female æt. 26, suffering from acute general miliary tuberculosis. Cæsarean section was performed during death agony; the child died in two hours. Tubercle was found in the placenta and in the fœtal placental villi.

---

[1] *Beiträge zur path. Anat.* vol. ix. p. 432; vol. xvi. p. 313.

Case III.—A woman æt. 25 died during pregnancy from acute miliary tuberculosis. Tubercle was found in the placenta and fœtal placental villi. Tubercle bacilli were found, on microscopical examination, in the liver of the fœtus and in a neighbouring lymphatic gland. There were in this case no macroscopic signs of tubercle in the fœtus.

Case IV.—A pregnant woman æt. 33 suffering from pulmonary tuberculosis, died from hæmoptysis. Cæsarean section was performed; the child was dead. Tubercle was present in the placenta and in the fœtal placental villi, but not in the body of the fœtus.

The authors suggest that the reason why so few bacilli pass into the fœtal circulation is that the changes in the vessels of the tuberculous villi lead to the formation of hyaline thrombi, which, with the overgrowth of the vascular endothelium, constitute a barrier to their passage.

3. Hochsinger has recently shown [1] that the virus of tuberculosis and syphilis may be jointly transmitted from parent to offspring.

In three infants the subjects of congenital syphilis, and presenting signs of pulmonary disease, this was found post mortem to be due to tuberculosis and not to syphilis. Tubercle bacilli were found in the lungs in all three cases. In two of the cases the mothers were known to be suffering from tubercular disease of the lungs. These cases are fully narrated in the chapter on Pulmonary Syphilis (*vide* p. 432).

The first case was observed in 1891 in a child not quite three weeks old; the second in 1891 in a child twenty-four days old; the third in 1893 in a child eleven weeks old.

The first and third of the cases described by Hochsinger may without much doubt be considered examples of congenital tuberculosis; the second is doubtful. In the first case, the fact that the child suffered from dyspnœa from the time of its birth, and that in the third week there were signs of great enlargement of the liver and spleen, which were shown by post-mortem examination to be due to a tubercular focus which had become caseous, render it extremely unlikely that the infection was from without.

It may therefore be considered as fully established that it is possible for a tuberculous mother to infect her child, but it is a long step from this to the acceptance of Baumgarten's view that this is the common mode of infection in tuberculosis. All experimental evidence is against the assumption that the growing tissues of young animals possess a special resisting power to the virus of tubercle. It is clear, however, that mere naked-eye examination of a fœtus for tubercle is insufficient. Microscopical examination, particularly of the liver, is essential, and the absence of results from inoculation is a still more important piece of evidence.

The writer is disposed, however, to believe that in the future evidence will accumulate in favour of the view that in some cases of tuberculosis of the glands, bones, and joints in children the virus has been directly transmitted from the parent, and has remained latent in the tissues of the child. Many such cases are difficult to explain on the assumption that infection is from without.

---

[1] *Wiener Medicinisch. Blätt.*, Nos. 20, 21 (1894).

But before such an explanation could be accepted in any given case it would be necessary to prove that the mother was tuberculous, and that the child had never been exposed to infection, particularly from tuberculous milk.

CONDITIONS PREDISPOSING TO PULMONARY TUBERCULOSIS

Heredity.—The influence of heredity may be shown by (*a*) the direct transmission from parent to offspring of a special liability to the disease, or by (*b*) the existence in certain families of a predisposition to the disease, the parents being either certainly or apparently unaffected.

Attempts have of late been made to discredit the generally accepted view that parents may transmit to their offspring a special liability to pulmonary tuberculosis.

It is contended that what is transmitted is a general predisposition to disease which belongs to the offspring of weakly parents, and that the frequent occurrence of successive cases of tubercular disease of the lungs in the same family is due to infection in the home. The question is admittedly a difficult one, and now that the real nature of the disease is understood the belief in the influence of infection in its etiology is in the ascendant and that of heredity has diminished. This is as it should be; but a belief in the importance of the hereditary factor is founded on the experience of centuries of observation and is not to be lightly got rid of, especially when it is held by those who have had unusual opportunities of studying the disease.

The line of argument by which it is sought to prove that the inherited predisposition is to disease in general and not especially to tuberculosis, usually starts by an inquiry into the family history of subjects of the disease, their statements as to the cause of death of their parents being accepted without so much of additional proof as would be obtained by the production of the certificate of death. Some experience of the work of a Life Assurance Office will enable anyone to appreciate the value of such evidence.

In cases in which a parent has died at an early age, if the death certificate is obtained, nothing is more common than to find the statement of the proposer at variance with it, and that death attributed to pneumonia or some other cause has been really due to tuberculosis.

On the other hand it is possible, if evidence of the kind above described is accepted, that deaths may be attributed to tuberculosis which were really due to some other cause; but the great objection which exists to an admission of a tubercular taint in a family tends to diminish this source of error. If, however, it is admitted that these two sources of error may balance each other, their existence renders evidence of the kind quite untrustworthy as a basis for inductive proof.

If it is sought by this method to ascertain the incidence of pulmonary tuberculosis in the children of non-tubercular and tubercular parents respectively, the inquiry must not start from a person the subject of the disease, as many families are, so far as can be determined during life, free from it altogether. To determine this point it would obviously be necessary to take a very large number of families without any kind of selection. But in order to settle the question even with regard to a single generation with scientific accuracy, the parents and every member of the family must be dead, and the cause of death must in every case have been determined by post-mortem examination.

Every pathologist of experience is aware that to find tubercular lesions where none were suspected is a matter of everyday occurrence in the post-mortem room, so common indeed that it is impossible to be certain whether an individual was or was not the subject of tuberculosis without an autopsy.

The mere statement of the conditions necessary renders it obvious that it is a profitless labour to attempt to settle this question by the methods generally adopted.

Many writers have furnished tables compiled from the statements of patients as to the cause of death of their parents and relatives, with a view to determine the effect of heredity, but the percentages obtained vary widely. In 12,509 cases collected by Dr. J. E. Squire from various sources, one or both parents are said to have been tuberculous in 3,101 instances, or 24·79 per cent.[1] In 12,146 cases, in which grandparents and collaterals are included, the influence of heredity appears in 62·34 per cent.

An analysis of 1,000 cases observed in private practice by Dr. Theodore Williams shows that in 12 per cent. a direct hereditary predisposition, *i.e.* from parent to offspring, was present, and in 48 per cent. there was a predisposition to the disease in the family. Dr. Wilson Fox found a history of direct heredity in 33 per cent. in hospital cases.

The most important effects of direct heredity (*i.e.* from the parents) are that the disease appears at an earlier age, especially in females (Williams), and runs a more acute course. The influence of the mother is almost certainly greater than that of the father as regards the children of both sexes.

The most remarkable case of family predisposition which has come under the notice of the writer is the following:

The information was obtained from a healthy-looking man æt. 52. His father was a tall thin man of rather delicate appearance, who lived a country life and rarely suffered from illness. He died at the age of 71 from 'senile decay.' His mother was a stoutly built woman, who died at the age of 63. The cause of her death is doubtful, but it was not due to 'consumption.' The informant has one brother, aged 59, living and in good health; he has no sisters living.

---

[1] *Med.-Chir. Trans.*, vol. 78, Dr. J. E. Squire.

The following table gives the causes of death, and the age at death, of his brothers and sisters :

	Age	Cause
Brothers dead:	10	Scarlet fever.
	16	All died from 'consumption.'
	19	
	20	
	24	
	36	
Sisters dead:	9	All died from 'consumption.'
	12	
	13	
	16	
	18	

All the sisters and three of the brothers died in the same village, but not in the same house. The family was well-to-do and inhabited a large house situated on high ground in a healthy neighbourhood in the south of England. After the death of two children the parents built a new house at a short distance from the old one.

General conditions inducing loss of resisting power.—We have already stated our belief that the susceptibility to tuberculosis is universal amongst mankind, and that the most robust health would not confer immunity provided the dose of the virus were sufficiently large, such, for example, as could be administered by repeated subcutaneous injections. It is probable also that the resisting power of almost any individual may be so reduced by unfavourable conditions as to render him liable to the disease as it is ordinarily acquired, in spite of the most complete freedom from hereditary predisposition.

Any condition which impairs the general health may therefore become a predisposing cause of pulmonary tuberculosis.

Insanitary conditions of all kinds, particularly the absence of fresh air and sunlight from dwellings and workrooms, are powerful predisposing causes of the disease.

Dwellers in large towns are more prone to tuberculosis than those who live in country districts; this is doubtless due to a variety of causes, amongst which density of the population, destitution, unhealthy occupations, breathing vitiated air, facility for infection, and the general depressing effect of life under such conditions, are the most important.

The effect of *chronic alcoholism* has been doubted, but recent statistics point clearly to its influence in predisposing to the disease.

Frequent child-bearing and *prolonged lactation*, the latter practice being often adopted with a view to delay pregnancy, are specially harmful to young mothers, and are often followed by tubercular disease of the lungs. The mother rarely dies during the period of gestation, but after delivery the disease often runs a very rapid course.

Occupation is an important factor in the etiology of the disease. Its marked prevalence amongst those who work in a dusty atmosphere has been dwelt upon in the chapter on disease due to the inhalation of dirt.

The conditions of work, and especially the absence of free ventilation in workshops or mines, are depressing to the general health, whilst the presence of dust in the atmosphere excites catarrh of the bronchi, and so induces changes which favour the lodgment and growth of the bacillus. Under such circumstances infection is extremely likely to occur.

Race and climate.—No race is free from the liability to tuberculosis, and although in certain areas the pulmonary variety of the disease is of rare occurrence, this depends apparently upon the conditions of life, and not upon racial immunity.

The climatic influences which favour its development are warmth and moisture and defective subsoil drainage; the two former are met with in subtropical climates at a low elevation, where the prevalence of the disease is very marked. The influence of the latter, which was first pointed out by Bowditch and Buchanan, has been confirmed by the subsequent observations of Thorne Thorne, who has shown that with improved drainage the prevalence of the disease is diminished.

The influence of *altitude* is illustrated by the rare occurrence of the disease in the Hartz Mountains (1,600 ft.) and the Black Forest, and at greater elevations in the Pyrenees (2,800 ft.) and in Switzerland (4,000 ft.). In southern latitudes a similar effect is not observed until a much greater height (7,000 to 8,000 ft.) is attained.

Various explanations have been offered of the comparative absence of the disease under such conditions, but it is highly probable that purity of the air, sparseness of population, the absence of insanitary conditions and, to a great degree, of the ordinary sources of infection are factors of much greater importance than mere elevation above the sea-level, and that if in all these respects the circumstances could be experimentally reversed the comparative immunity would disappear.

Age and sex.—The following table, which is taken from Dr. Wilson Fox's work,[1] was compiled by Dr. W. Ogle, Registrar-General.

Mean Annual Mortality from 'Phthisis' per Million living at all Ages and at twelve successive Age-periods

Period, 1851–80	All ages	0–	5–	10–	15–	20–	25–	35–	45–	55–	65–	75 and upwards
Males .	2,418	1,034	432	616	2,088	3,676	3,941	4,097	3,850	3,274	2,112	730
Females	2,428	993	491	1,061	3,008	3,798	4,165	3,826	2,812	2,075	1,322	523

---

[1] *Op. cit.* p. 520.

All forms of 'phthisis' are included in this table, and probably a number of cases which would have proved on autopsy not to be tubercular. On the other hand, it is certain that a very much larger number of cases of pulmonary tuberculosis are excluded owing to erroneous diagnosis.

The above table shows that in this country,

(*a*) If all ages be included, there is practically no difference between the two sexes as regards liability to death from 'phthisis.'

(*b*) In the first five years of life the death rates are nearly the same for the two sexes.

(*c*) Between the ends of the fifth and thirty-fifth years the death rate amongst females is greatly in excess of the male rate.

(*d*) After the thirty-fifth year the male rate is higher than the female. This may possibly be due to the fact that the liability of females being in part dependent upon defects of resisting power induced by child-bearing and lactation, those most susceptible to the disease have been carried off during the earlier period.

(*e*) The number of deaths from 'phthisis' at advanced ages is very considerable. (A fact which must have impressed everyone who has had an extended pathological experience.)

Dr. Ogle shows in other tables that the differences between the male and female death rates are special to 'phthisis' (pulmonary tuberculosis) and do not exist in the mortality from tubercular disease of other organs, in which the male death rate is in excess of the female at all ages.

Previous disease.—*Pleurisy.*—Not infrequently, but in exactly what percentage of cases it is difficult to state, an attack of acute pleurisy precedes the appearance of tubercular disease of the lungs. Various explanations have been offered as to the exact relation between the two affections:

1. The pleurisy may be a primary manifestation of tuberculosis, and the lungs may be secondarily infected.

2. The disease of the pleura may be secondary to tubercular disease already present in the lungs.

3. The impaired functional activity of the lung resulting from the attack of pleurisy may act as a predisposing cause of tuberculosis. The fact, however, that the subsequent tubercular disease very often appears first in the opposite lung is opposed to this view.

4. The pleurisy may be non-tubercular and the first evidence of a general lack of resisting power to disease, the existence of which is confirmed by the subsequent occurrence of tubercle of the lungs.

It is not necessary to suppose that one and one only of these theories is applicable to all cases, but in considering the etiology of pleurisy (*vide* p. 545), strong evidence is adduced in favour of the view that in the majority of such cases the attack of pleurisy is due to an acute tubercular infection of the serous membrane.

Absolute proof of this is difficult to obtain, as recovery is the rule;

but that such a condition may occur is within our own experience, as the writer has made a post-mortem examination on a case of acute pleurisy terminating in empyema, and submitted to operation, which proved to be due to acute tuberculosis of the serous membrane. There was no trace of tubercle in the lungs or any other organ of the body, except possibly a bronchial gland.

Bronchial catarrh.—The view that pulmonary tuberculosis may originate in a 'neglected cold' was at one time generally accepted, and is still firmly held by the laity. In a considerable proportion of such cases, it is at any rate highly probable that the disease has been tuberculous from the first, but it cannot be denied that bronchial catarrh may prepare the way for tubercular infection, and also that it is common amongst the subjects of that disease. In many cases, however, previous to the development of pulmonary tuberculosis, and subsequently, there is no special liability to catarrh.

Having regard to the extreme prevalence of catarrhal affections of the bronchi, and the relatively small number of cases in which tuberculosis follows, we are disposed to think that the importance of this factor in the etiology of the disease has been exaggerated.

Pneumonia.—There is a general agreement that an attack of pneumonia is rarely followed by tuberculosis, and many cases supposed to have this origin are really examples of the lobar type of caseous tuberculosis.

Disease of the cardiac valves.—The subjects of congenital stenosis of the pulmonary orifice, who attain to adult age, not uncommonly develop tubercular disease of the lungs. Our experience has led us to the belief that the antagonism between affections of the mitral valve and tuberculosis of the lungs is less absolute than was supposed by Louis, who first formulated that theory. In a demonstration of cases of valvular disease given by the writer at the Brompton Hospital some years ago, he was able to show from among his out-patients attending at the time four cases of mitral stenosis associated with pulmonary tuberculosis.

Diabetes.—Pulmonary complications, such as pneumonia and gangrene, are not uncommon in the course of diabetes, but a still closer relationship exists between that disease and tuberculosis of the lungs, nearly 50 per cent. of cases of diabetes, according to Griesinger's statistics, presenting tubercular lesions. Diabetic 'phthisis' was regarded as a special variety of the disease, but the proof that tubercle bacilli are invariably present either in the sputum or lungs has been fatal to that as to many other varieties of 'phthisis.' The pulmonary complication does not, as a rule, appear until the disease has lasted a year or two, and is apt to be of the caseous type; but it may undergo arrest.

Cancer.—It is very rare indeed to find tuberculosis and cancer in active progress at the same time, but such cases have been observed. It is, however, extremely common to meet with evidence of arrested tuberculosis of the lungs after death from cancer.

Of 177 cases of obsolete tubercle of the lungs observed by the writer[1] at the Middlesex Hospital between 1879–86, in 73 cases, or 41·2 per cent., the cause of death was cancer.

Mr. Roger Williams[2] has shown that it is more common in the subjects of cancer to find a family history of tuberculosis than of malignant disease. Cancer would appear, therefore, to specially affect such offspring of the tuberculous as escape the disease of their parents.

It is certainly remarkable that the tissues of an individual who ultimately succumbs to a disease of the type of cancer should have had at one period resisting power sufficient to overcome an established tuberculosis of the lungs. When we know more of the origin of cancer this apparent anomaly may possibly be explained.

Syphilis.—The relation of tuberculosis to syphilis is considered in the chapter on Pulmonary Syphilis (*vide* p. 429).

Many other affections have a more or less indirect relation to pulmonary tuberculosis, but of such it can only be said that by lowering the health of the individual they diminish his resisting power, and so render him more liable to disease.

J. K. F.

---

[1] *Arrested Pulmonary Tuberculosis.*

[2] *Middlesex Hospital Reports.*

CHAPTER XXVIII

PULMONARY TUBERCULOSIS

(continued)

THE VARIETIES OF PULMONARY TUBERCULOSIS

	PAGE
Miliary tuberculosis	319
Caseous ,,	320
Fibroid ,,	320
Chronic or fibro-caseous tuberculosis	322

THE difficulties which attend the use of 'phthisis,' as a generic term, are trifling compared with those which await any writer who may attempt, under that heading, to describe the varieties of the disease.

Anyone engaged in medical teaching must have witnessed the hopeless nature of the attempt by a conscientious student to differentiate catarrhal phthisis from tuberculous phthisis, or pneumonic phthisis, whether acute, chronic, confluent, or disseminated, from the tuberculous or tuberculo-pneumonic variety. This list is by no means complete, for it does not include fibroid, bronchiectatic, diabetic, miner's, collier's, knife-grinder's, potter's, or syphilitic phthisis; or acute tuberculosis of the lungs. The latter affection has, in truth, always been regarded as a separate disease, and relegated to a different category, whereby its close relationship with the other forms of tuberculous disease has been necessarily obscured.

It must be confessed that when one was in the habit of using the term 'phthisis,' a feeling of wondering admiration was experienced for those who could distinguish fifteen varieties of a disease, of which one could with difficulty recognise but three or four.

It is now obvious that no such number of well-marked varieties of this disease as is above enumerated really exists, and it is satisfactory to know that many of the terms mentioned have already gone out of common use. There is, however, another danger ahead, for we are threatened with the terms 'bacillary' and 'non-bacillary phthisis,' each of which may, as time goes on, be still further subdivided.

The **classification** of the various forms of the disease which is adopted in this work is as follows:

(1) Miliary tuberculosis of the lungs.
(2) Caseous tuberculosis of the lungs.
(3) Fibroid tuberculosis of the lungs.
(4) Fibro-caseous or chronic tuberculosis of the lungs.

The pathological features of tubercular lesions, upon which this classification is based, depend upon and are at the same time a measure of the activity of the morbid process.

The use of this classification is of very great value in attempting to forecast the course of the disease, because we are able to associate certain clinical features, and *particularly the occurrence of well-marked types of pyrexia*, with the presence of definite pathological changes in the lungs.

Having now made use of this classification for some years, the writer is able to state that others have also found it of service, and that students experience but little difficulty in recognising well-marked examples of the conditions which it differentiates.

It is true that, owing to changes which may occur from time to time in the rate of progress of the disease, the same case may at different periods present the features characteristic of different varieties of the affection; but this is an objection, which applies equally to all classifications hitherto suggested, and does not seriously impair the value of that here adopted, as, although the morbid process may alter in character, it is of great importance to be able to recognise the form which is present when the patient is actually under observation.

It will be useful, as a preliminary to a detailed consideration of the various forms of pulmonary tuberculosis, to give a brief summary of their pathological and clinical characters.

MILIARY TUBERCULOSIS OF THE LUNGS

This is marked by a rapid formation of miliary tubercles throughout the lungs, and it may occur when they were previously free from tubercular disease, or as a terminal event in cases of the chronic variety, or from the breaking down of an encapsuled caseous lesion of limited extent, the result of long past and arrested disease.

When, as often happens, especially in children, many other organs of the body are similarly affected at the same time, the clinical aspect of the case is chiefly influenced by the presence or absence of infection of the meninges of the brain. If they are involved, it assumes the characters of tubercular meningitis, and the pulmonary symptoms may be more or less completely masked. The occurrence of acute miliary infection of the lungs in the course of chronic pulmonary tuberculosis may be recognised by the appearance of pyrexia of a peculiar type (see p. 325).

CASEOUS TUBERCULOSIS OF THE LUNGS

This variety of the disease corresponds to that variously termed 'acute phthisis,' 'acute pneumonic phthisis,' 'tuberculo-pneumonic phthisis,' 'florid phthisis,' 'galloping consumption,' 'broncho-pneumonic phthisis,' 'scrofulous phthisis,' 'epithelial pneumonia,' &c. &c.—a truly formidable list of names.

The miliary granulation forms a comparatively unimportant feature in the pathological changes, which are specially characterised by the rapidity with which the areas involved in the tuberculous process undergo caseation. This is in inverse ratio to the resisting power of the individual. Such cases are marked by a rapid course, which may be continuously downward, or, as more often happens, there are from time to time periods during which the activity of the morbid process is lessened.

An important and special feature is that nearly all the structures of the lung in the affected area are involved in the morbid process. This form of the disease is also accompanied by pyrexiæ of a characteristic type (*vide* p. 327).

Cases are often observed intermediate in severity between those just referred to and such as are classified as examples of chronic pulmonary tuberculosis. They are characterised by the same tendency to caseation of the lesions, but the progress of the destructive changes throughout the lungs is less rapid. At the outset the symptoms may be acute, but after possibly the upper lobe of one lung has been extensively involved and softening has taken place, the elimination of a caseous mass is followed by a remission of the fever, and the patient begins to improve.

When a case is seen after this favourable change has occurred, it may be recognised as belonging to this class by a comparison between the brief duration of the illness and the wide extent and destructive character of the lesions.

FIBROID TUBERCULOSIS OF THE LUNGS

This form presents in every particular the most complete contrast to that just described. There may be, indeed, such an absence of symptoms and physical signs as to make it appear doubtful whether the case is really one of tuberculous disease of the lungs at all.

Its distinctive pathological feature is the fibroid transformation of miliary tubercles, which do not reach the stage of either caseation or softening.

We desire especially to direct attention to the differences between this variety of tuberculosis and the disease to which the term 'fibroid phthisis' was applied by the late Sir Andrew Clark. His description of that disease is briefly as follows:[1]

'The general symptoms, viewed collectively, are strikingly different from those of tubercular phthisis. . . . In fibroid phthisis there is always contraction of the chest wall. The percussion dulness is harder, higher-pitched, and more uniformly continuous than in any other lung disease. Resistance of the thoracic parietes is greatly increased. Intercostal spaces are depressed. Sometimes the dulness is tubular. Vocal fremitus is at one time greatly increased, at another diminished. Over the fibroid lung one hears blowing breath sounds, often without audible prolongation of expiration; occasionally coarse, dry and moist râles; superficial creaking, and diffuse bronchophony; over cheesy deposits of any extent the breath sounds are sharply tubular, the expiration prolonged, and the vocal resonance bronchophonic, sniffling, circumscribed.'

'The disease is commonly slow in its progress, and when it complicates tubercles it retards disintegration and greatly prolongs life.'

Although the term 'fibroid phthisis' had previously been used by Laycock and H. G. Sutton, it is to Sir Andrew Clark that we are chiefly indebted for the recognition of cases of the kind he described. In some respects, however, the conclusions which he drew as to the pathology of such cases were, in the writer's opinon, erroneous —chiefly the belief in their non-tuberculous nature, and also as to the part played by the pleura in the production of the 'fibrous bands running through the contracted lung.'

As the late Dr. Moxon said, these are only cases of 'phthisis of which the history has been forgotten,' owing to their very chronic course. In many cases of less prolonged duration the upper lobes of the lung present the most typical features of 'fibroid phthisis,' whilst in the lower lobes the tubercular nature of the lesions is obvious. If the patient lives until the same changes have taken place in the lower lobes, the case becomes one of 'fibroid phthisis.'

In the writer's opinion the thickening of the pleura follows the contraction of the lung, and the fibrous bands are generally thickened interlobular septa, and are only rarely due to ingrowths from the surface of the pleura.

Cases which we should admit to be examples of fibroid tuberculosis of the lungs are, it is true, included in the description of the disease given by Sir Andrew Clark, but there can be no doubt that the term 'fibroid phthisis' has practically become restricted to cases of the type pictured in the extract just quoted—that is, to cases in which there is marked retraction of the chest, absolute dulness on percussion, and blowing breath sounds—probably because their recognition is comparatively easy; whereas in fibroid tuberculosis there may be extensive disease with but few signs to indicate its presence, and instead of dulness there may be hyper-resonance from emphysema.

CHRONIC OR FIBRO-CASEOUS PULMONARY TUBERCULOSIS

In this, the common form of the disease, the lesions indicate that at various periods the morbid process has been active, but that from time to time the resisting power of the tissues has been able to assert itself and to check the advance of the disease. Cavities are formed either by the softening of areas of caseation or by a slower process of disintegration, but inflammatory changes at their margins have led to the production of a fibrous wall, by which the farther extension of the disease has been prevented. Caseous areas are found to be surrounded by fibrous capsules or transformed into calcareous masses, and miliary tubercles have undergone fibrosis.

The degree of resisting power has not been maintained throughout, as in the fibroid form of the disease, but at times it may have been sufficient to produce complete arrest of the process, and it has only at rare periods been so low as to permit of the rapid caseation of considerable areas. Towards the close of life, however, either this change or an acute miliary tuberculosis of the parts of the lungs, until then unaffected, not uncommonly occurs.

These phases of the morbid process give rise, during the prolonged course of a chronic case, to symptoms of varying severity similar to those which characterise the more and less acute forms of the disease.

J. K. F.

CHAPTER XXIX

PULMONARY TUBERCULOSIS

(continued)

PAGE
Pyrexia in miliary tuberculosis 325
„ „ caseous „ 327
Temperature in fibroid „ 328
Pyrexia in chronic or fibro-caseous tuberculosis 329

PYREXIA IN PULMONARY TUBERCULOSIS

Some years ago the writer[1] called attention to the fact that *by observation of the temperature in a case of pulmonary tuberculosis it is possible to determine the nature of the changes in progress in the lungs*, and subsequent experience has confirmed the correctness of the views he then expressed. The miliary, caseous, and fibroid varieties of the disease are characterised in each case by a definite and distinct range of temperature, and this is present both in typical examples of those forms of the disease and also when, as frequently happens, they appear as temporary complications during the course of a case of chronic pulmonary tuberculosis.

In order to render as clear as possible at the outset the features by which each variety of the disease may be recognised, we shall now consider the characters of the accompanying pyrexia. Before doing so, however, something may be said as to the daily range of temperature often met with in tubercular disease. Pyrexia commences or increases in the afternoon, often about 2 P.M.; the temperature gradually rises, and attains its maximum between 6 and 10 P.M., the exact time varying in different cases. A continuous decline then occurs until 4 or 5 A.M., when in some cases the thermometer may register a temperature of one, two, or even three degrees below the normal. A temperature so low as 91·6° F. has even been recorded. This fall is followed by a recovery of body heat, and at 10 or 11 A.M. the temperature may in such cases be normal.

This type of pyrexia is not, however, peculiar to tubercular disease, although it certainly occurs therein with especial frequency; it is an exaggeration of the normal daily range of the body temperature.

When the daily range of temperature in a case of pulmonary tuberculosis accompanied by rapid disintegration of the lung is marked by an extreme difference between the highest and lowest points ('hectic type'), the fever is generally ascribed to septic infection. Koch has recently stated[1] that a temperature over 100·4° in a case of pulmonary tuberculosis implies the presence of septic organisms, and contra-indicates the use of his new tuberculin product. Other writers also profess to be able in such cases to distinguish between the pyrexia due to specific and septic infection respectively, and base their classification of cases suitable for certain kinds of climatic treatment on the presence or absence of septic fever. It is clear therefore that this question of the cause of the fever in tuberculosis is one of great importance. The views which the writer has long held on this point are somewhat at variance with those just stated.

When fever arises from 'septic infection' the condition present is septicæmia, a disease accompanied by certain lesions—and particularly by enlargement of the spleen—which have not, so far as we are aware, been shown to occur in cases of pulmonary tuberculosis characterised by pyrexia of this type.

Pyrexia of this character may be observed in its most typical form in cases of tubercular pleurisy or peritonitis, where there is no evidence of septic infection. In a case of double pleurisy, almost certainly of tuberculous origin, under the care of the writer in the Middlesex Hospital, a 'hectic' temperature persisted for nearly eleven weeks, suggesting at first the presence of an empyema, a suspicion which was twice negatived by the withdrawal of a clear serous fluid from the chest.

The conclusions to which we have been led by a comparison of the temperature charts in fatal cases of the disease with the lesions found post mortem are, speaking generally, as follows:

(1) The degree of fever present in cases of pulmonary tuberculosis is a measure of the activity of the morbid process.

(2) Marked pyrexia is always associated with an increase in the tubercular lesions.

(3) The action of the septic organisms present in the lung has little or no share in its production.

(4) The occurrence of miliary, fibroid, and caseous lesions is indicated by distinct types of pyrexia.

It is perhaps scarcely necessary to add that the subjects of tuberculosis are, like other individuals, liable to fever from various causes, and that the foregoing statements do not imply a belief that pyrexia in such cases is always due to an extension of the disease present in the lungs.

---

[1] *Deutsch. med. Wochenschr.*, April 1, 1897.

The pyrexia is most marked when the disease is making rapid progress in an organism still capable of reaction. This latter condition is essential, as in cases characterised by considerable failure of vital power, continuous extension of the disease, leading up to a fatal termination, is not uncommonly observed in association with an absence of pyrexia.

PYREXIA IN MILIARY TUBERCULOSIS

Miliary tuberculosis of the lungs may, as already stated, occur as part of a general infection, or as a terminal event in a case of the chronic form of the disease.

It is characterised by two distinct varieties of pyrexia, (*a*) the continuous and (*b*) the inverse.

(*a*) The continuous type is represented in the accompanying chart from a case of chronic tuberculosis terminating with an acute infiltration of miliary tubercles throughout the remaining unaffected portions of the lungs. When in a case of this kind an infiltration of miliary tubercle is in progress, the temperature usually rises gradually for a few days and then continues high, with very slight morning remissions. In this case it ranged between 101° and 103°, but it may fluctuate at a still higher level.

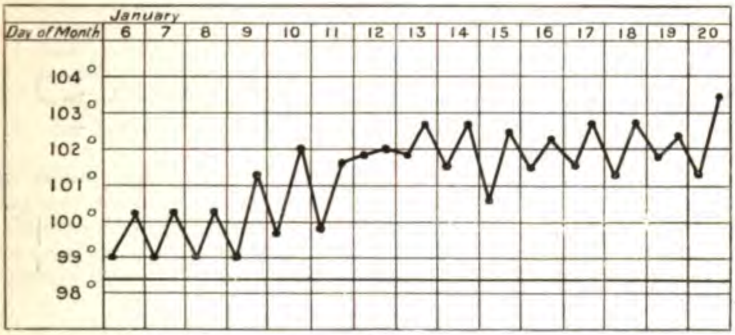


Fig. 87.—Continuous Type of Pyrexia in Miliary Tuberculosis

(*b*) The 'inverse' type of temperature is characterised by the fact that the pyrexia attains its maximum in the morning instead of the evening. This may be met with in cases of pulmonary tuberculosis previously of the chronic variety, and also in cases of general acute tuberculosis, and may then be the only trustworthy sign of the nature of the disease.

Reference may be made to a case of the latter nature recorded by Dr. D. W. Finlay in vol. xxii. of the 'Transactions of the Clinical Society of London.'

The accompanying chart (fig. 88) is taken from a case of chronic tuberculosis under the care of the writer in the Brompton Hospital in which the lungs, except in such parts as were not the site of old lesions, were found after death to be infiltrated with miliary tubercles, chiefly of recent formation. The temperature curve in this type of pyrexia differs from that just described, not only in the reversal of the period of the day in which the maximum of fever occurs, but also in the occurrence of more marked remissions or of short intermissions. Death may in such cases be preceded for some days by a fall of temperature, but the inverse type may still be maintained.

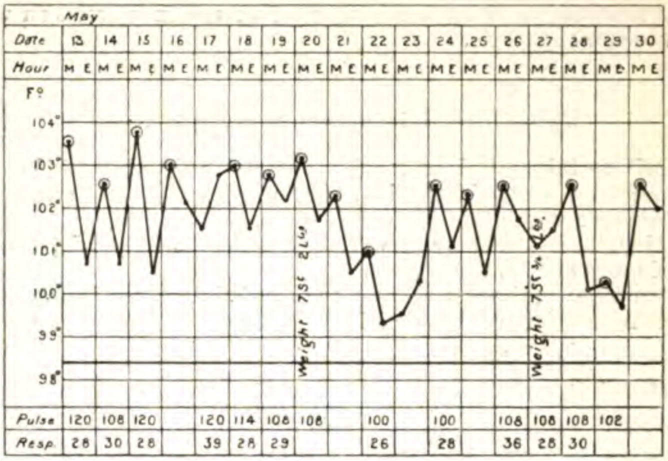


FIG. 88.—INVERSE TYPE OF PYREXIA IN MILIARY TUBERCULOSIS

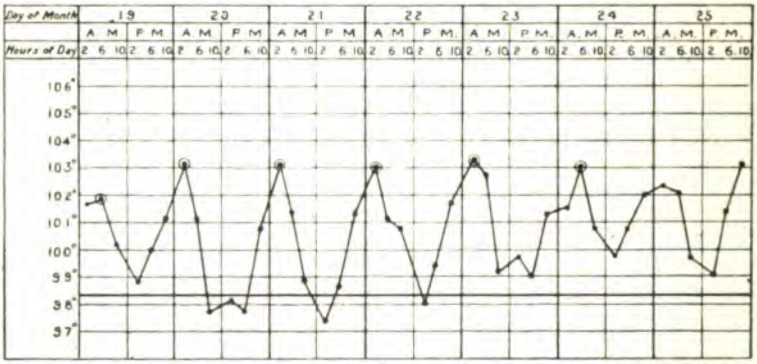


FIG. 89.—INVERSE TYPE OF PYREXIA IN MILIARY TUBERCULOSIS

The accompanying chart (fig. 89) shows that this type of fever may be produced by the evening rise of temperature being continued into the early hours of the morning (2 to 6 A.M.).

The importance of the inverse type of temperature as a guide to prognosis is considered on page 381.

PYREXIA IN CASEOUS TUBERCULOSIS

This variety of the disease is marked by a high remittent type of pyrexia, and in the most acute cases almost throughout their course.

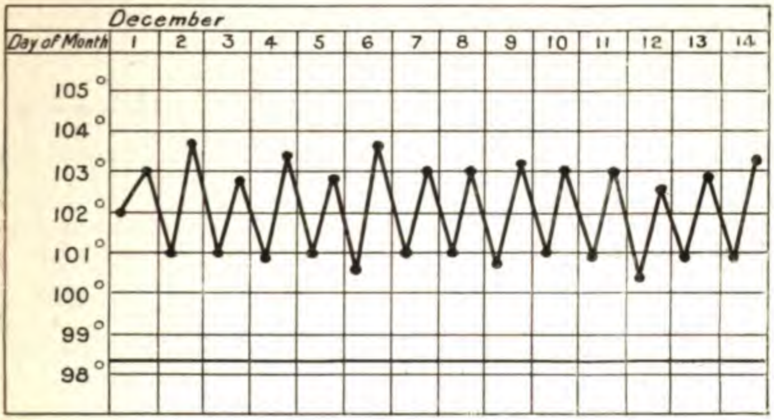


Fig. 90.—Pyrexia in Caseous Tuberculosis

The temperature ranges between 101° and 104° (*vide* fig. 90), the remissions being as a rule much more marked than in cases of miliary tuberculosis.

The following chart (fig. 91) is from a case which commenced with extremely acute symptoms; and illustrates the type of pyrexia at the onset of an attack of tuberculosis which afterwards assumed the caseous form. The disease, after a duration of about eighteen months, finally underwent arrest.

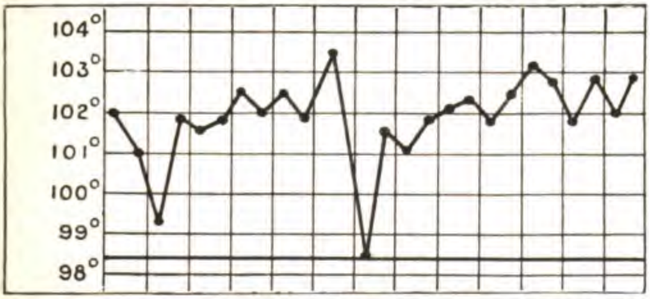


Fig. 91.—Chart for the first fortnight from a case of Caseous Tuberculosis, in which the Disease subsequently underwent arrest

At a later stage, when cavities have formed in both upper lobes and active softening is in progress in the lower lobes, and vital power is diminishing, the fever tends to assume the intermittent type illustrated in fig. 92, a very marked rise in the evening to 102° or 103° F., or even higher, being followed by a fall in the early morning to 97° or 96° F.

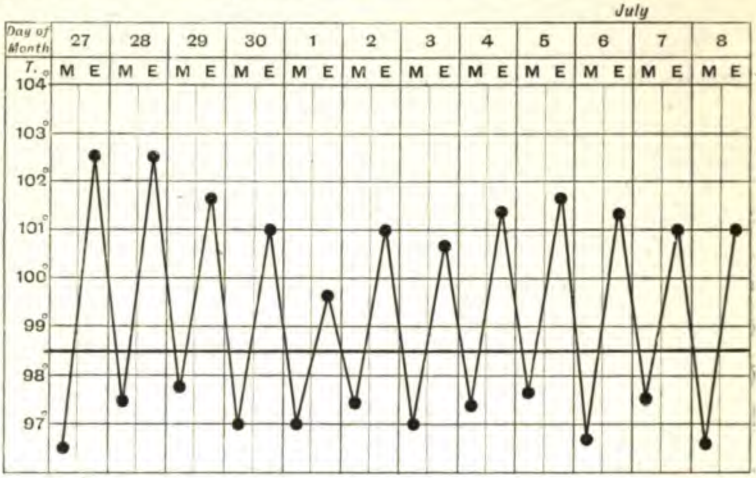


Fig. 92.—Pyrexia in Caseous Tuberculosis with failure of Vital Power

TEMPERATURE IN FIBROID TUBERCULOSIS

This variety of the disease is characterised by a more or less complete absence of fever (*vide* fig. 93). Often for periods of prolonged duration the temperature may not be above normal, and for several days in succession it may be subnormal.

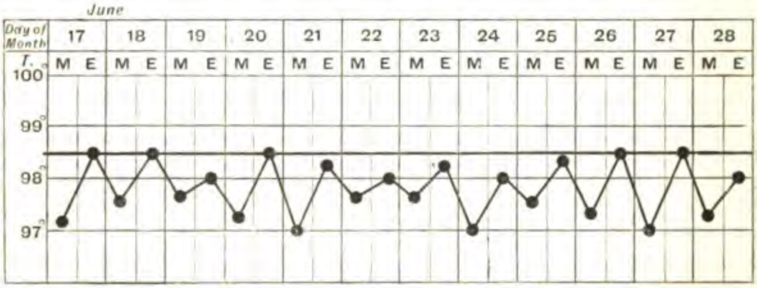


Fig. 93.—Chart from a case of Fibroid Tuberculosis

In some cases a moderate evening rise, perhaps to 99° F., may be observed to occur occasionally.

PYREXIA IN CHRONIC OR FIBRO-CASEOUS TUBERCULOSIS

The temperature present during the prolonged course of a case of chronic tuberculosis is marked by great variety of range, and for long periods, during which the disease is quiescent or completely arrested, fever may be absent.

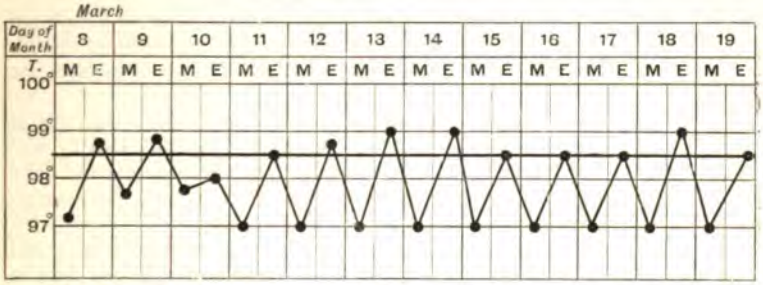


FIG. 94.—CHART FROM A CASE OF CHRONIC TUBERCULOSIS, QUIESCENT;

Periods of activity are accompanied by pyrexia in which the fever assumes the characters already described as peculiar to the various lesions.

The changes in the lung which immediately follow infection by the virus of tubercle and attend the formation of the initial

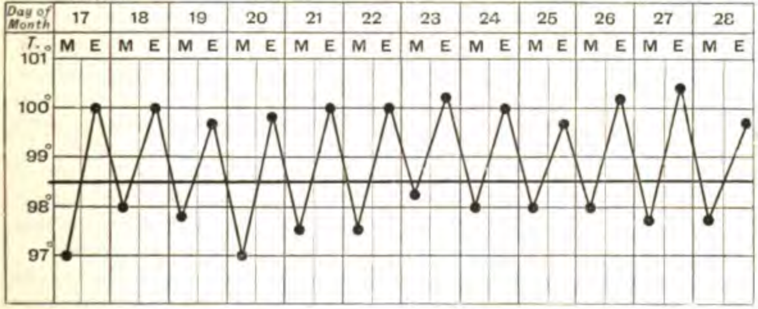


FIG. 95.—CHART FROM A CASE OF CHRONIC TUBERCULOSIS, INFILTRATION AND COMMENCING SOFTENING

lesion are probably accompanied by pyrexia, but it is not possible to make any definite statement upon this point.

It is very common indeed to meet with cases in an early stage of the disease presenting signs of infiltration limited to on apex, or possibly also involving the lower lobe of the same lung, in which fever is absent. It is possible, however, that the disease is then in a condition of arrest. A sudden increase in the area of lung affected, or an accession of activity of the morbid process without the presence of definite signs of extension, is almost invariably marked by the occurrence of fever.

As infiltration progresses and foci of softening make their appearance, about the apex or elsewhere, the temperature curve is apt to assume the form shown in fig. 95. The tuberculous type is maintained, but the evening rise is still moderate (100·5° or 101° F.), and the morning fall is distinctly marked.

It is often observed that with the formation of a well-defined cavity the pyrexia either subsides or disappears altogether. Such an event almost invariably marks the onset of a period of quiescence or complete arrest of the morbid process.

Speaking generally, the temperature in chronic tuberculosis of the lungs is characterised by great regularity; it may rise in the evening and fall in the morning almost to the same points for days or even weeks in succession, provided no marked change occurs in the activity of the morbid process.

The remarkable regularity of the evening rise and morning fall is liable to be disturbed by a number of mischances, some one of which is almost sure to happen sooner or later in any given case. For example, when a sudden increase in the evening fever is associated with a less marked morning remission, when on successive days the lowest point touched gradually recedes from the normal, and regularity is replaced by marked variability, we are rarely wrong in concluding that an extension of the morbid process has occurred. This may take the form of a rather rapid consolidation in the neighbourhood of the area of active change, but is more often due to a formation of miliary tubercle in parts of the lung hitherto unaffected. In attempting to determine from the character of the pyrexia the nature of the changes in progress in the lung, the observer is very likely to fall into error unless he is careful not to form an opinion without a knowledge of the morning and evening temperatures for at least a week. [In acute cases the temperature should be taken every four hours.]

Even this period may be too short when the surroundings of the patient have been recently changed; for example, the temperature during the four or five days following the admission of a patient into a hospital may give indications which are quite unreliable.

The temperature chart and the record of the patient's weight should always be considered together. If a low temperature or a diminution of fever is accompanied by a gain of weight, it is almost certain that the morbid process is at any rate inactive, and it may be in arrest. The significance of a low temperature which is accompanied by a loss of weight may be quite different.

J. K. F.

CHAPTER XXX

PULMONARY TUBERCULOSIS

(continued)

MILIARY TUBERCULOSIS OF THE LUNGS

	PAGE
Etiology	331
Morbid anatomy	332
Symptoms	333
Physical signs	334
Diagnosis	334
Prognosis	335
Treatment	335

Etiology.—It was first pointed out by Buhl that in the majority of cases of acute generalised tuberculosis, if careful search be made, a softening caseous mass will be discovered in some organ or tissue of the body. This was found in twenty-one out of twenty-three cases of the kind recorded by him. When the lungs are specially affected, it is generally a tubercular lesion which has broken down and discharged its virulent contents into a blood-vessel which, as pointed out by Weigert, is often a branch of the pulmonary vein; sometimes a caseous tracheal or bronchial gland is the source of infection.

Reference has already been made to the fact that many cases of chronic pulmonary tuberculosis terminate with an acute formation of miliary tubercle in the parts of the lungs hitherto free from disease.

The statistics of the writer,[1] and of Heitler,[2] giving the cause of death in cases presenting obsolete tubercular lesions of the lungs, show that in these cases also death often occurs from acute tuberculosis, and that these obsolete lesions are the source of infection. The risk of such an occurrence is very great when a caseous mass is present; it is decidedly less when tubercle has undergone fibroid transformation.

In a case recorded by the writer,[1] a caseous mass at the apex of the left lung, which had probably been encapsuled for forty years, broke down and caused death from acute pulmonary tuberculosis in twenty-eight days. There was an exactly similar mass firmly encapsuled at the apex of the right lung.

In some cases, however, no obvious source of infection is discovered post mortem. In a considerable proportion of these the disease has followed upon some acute fever, such as measles or whooping cough, and more rarely typhoid fever. It is possible that in such cases either a general lack of resisting power may have been induced by the previous disease, or the lungs may have been rendered specially susceptible by the associated catarrh or bronchopneumonia.

Miliary tuberculosis of the lungs is common in young children, and the lesions are then widely disseminated. It may, however, occur at any age, but is comparatively rare after 50. Of twenty-five cases observed at the Middlesex Hospital from 1884 to 1890 nineteen occurred in males and six in females; this preponderance of the male sex is in accord with general experience. In the above series an old caseous mass in the lungs was the source of infection in nine cases, a chronic cavity was present in five, an 'unresolved pneumonia infected by tubercle' in one, and in one case 'old pigmented scars' at the apex were the only obsolete tubercular lesions found. In one case in a child aged 5 years a caseous bronchial gland appeared to have been the primary focus; two were probably secondary to tubercular pleurisy, and in three infection spread from the lungs, but no old lesions were found there; in three no source of infection was discovered. In nineteen cases therefore out of the twenty-five the lungs were the original seat of the disease.

Morbid anatomy.—Miliary tubercles are usually found in great numbers throughout the lungs, and the appearance of the granulations is always suggestive of a recent formation. In children they are commonly of the grey, semitransparent character, varying in size from a pin's head to a pin's point, and may show no sign of caseation.

When the disease is secondary to chronic tuberculosis of the lungs in adults, some granulations may be of this nature, whilst others are rather less sharply defined and tend to run into groups, and although obviously of quite recent formation they are already beginning to caseate. In such cases they are always present in greatest number in the neighbourhood of the primary lesion, and gradually diminish in number towards the bases of the lungs. In children they are as a rule discrete, uniformly and thickly scattered throughout the organs, and the same appearances are observed in adults with miliary tuberculosis when many organs are affected.

The lungs are often large and intensely hyperæmic; the bronchi are inflamed and the bronchial glands enlarged, and generally in process of caseation. In many cases the pleura is extensively infiltrated with discrete miliary granulations. If the cerebral meninges are involved the infiltration may be widespread and present the ordinary appearances of tubercular meningitis, or death may occur when only a few very fine granulations have formed around the circle of Willis and at the inner extremity of the Sylvian fissure. In cases of generalised miliary tuberculosis the infiltration of the peritoneum may be extensive, but it is often limited to the under surface of the diaphragm, where a number of fine granulations may be found. They usually caseate very rapidly in this situation.

---

[1] *Proceedings of the Med.-Chir. Soc.*, series iii. vol. viii. p. 48.

Symptoms.—The onset may be either gradual or somewhat sudden, the former being the more common. The symptoms are those which usually attend an acute febrile illness, such as malaise, loss of appetite, headache and depression, but an initial rigor is rarely observed. The skin is generally moist and there may be free perspiration.

An attack of hæmoptysis may mark the onset of the acute illness when an old tubercular lesion is present in the lungs. In a child aged 3 years who died suddenly from profuse hæmoptysis, the writer found post mortem, in addition to a discrete miliary tuberculosis of the lungs, a recent caseous patch the size of a hazel nut at the anterior margin of the right lower lobe. This had softened in the centre, and the small cavity thus formed contained an aneurysm, the rupture of which was the cause of the hæmoptysis.

The pulmonary symptoms vary with the nature of the case. If the infiltration of the lungs is part of a general acute tuberculosis they may be but slightly marked and only suggestive of a moderately severe bronchial catarrh. Cough is usually present; it may be hacking and dry or accompanied by clear mucoid expectoration.

When the lungs are primarily or chiefly affected, cough is more severe and of the same character. Expectoration may be completely absent throughout the illness; if present, it may be mucoid or muco-purulent or streaked with blood; it is said occasionally to have the 'rusty' character of pneumonia, but this must be rare.

Dyspnœa is observed almost from the first, and as the case progresses the respirations become extraordinarily rapid, perhaps fifty or sixty per minute in an adult, and as many as eighty or even more in a child.

Cyanosis is a very important symptom of the disease; it appears as soon as the extent of lung affected is considerable, and gradually increases with the progress of the infiltration. It is most marked in the lips and the tips of the fingers, whilst the cheeks are flushed or of a slightly cyanotic tint.

Pyrexia is always present in the pulmonary type of the affection, although it may be absent or but slightly marked in general tuberculosis if the patient is extremely debilitated. The characters of the pyrexia have already been described (*vide* p. 325).

The pulse is feeble and extremely rapid. The spleen is generally enlarged, and if infiltrated it may be tender. Albuminuria may be present.

When, towards the end, the disease becomes widespread, the patient may fall into the so-called 'typhoid' condition, with dry brown tongue, low muttering delirium, and subsultus.

Physical signs.—If there is an old and well-marked pulmonary lesion it will be recognised by the ordinary signs; but in cases of arrested tuberculosis with a limited area of disease, close attention should be paid to signs observed on inspection and palpation of the upper part of the chest. A very slight defect in the expansion at one apex and a slight increase in the vocal fremitus beneath the clavicle may be the only signs indicating the presence of an old lesion.

Owing to the small size of the granulations and the way in which they are scattered through the lungs, the resonance of the percussion note may be normal or it may be only slightly impaired, or the note may be hyper-resonant from the over-distended state of the lungs.

Alterations in the note observed from day to day have been attributed to local collapse, followed by re-expansion (Eustace Smith).

On auscultation the respiratory sounds may be harsh and expiration prolonged, and fine râles may be heard, possibly only after cough. These are at first usually bubbling or faintly crackling in character, and may be accompanied by sibilant rhonchi. As the case progresses the râles may acquire a more decidedly crackling or crepitant character.

When miliary infiltration occurs as the terminal event in a case of chronic tuberculosis, there may be few signs, if any, in the previously unaffected portions of the lungs to indicate that a rapid formation of tubercle is in progress there.

The marked discrepancy between the physical signs and the general condition of the patient, and the presence of cyanosis and of the characteristic temperature, are then the most important guides to a correct diagnosis of the disease.

Diagnosis.—The ordinary proof of the existence of a tubercular lesion of the lungs—viz. the discovery in the sputa of tubercle bacilli—often fails in these cases. There may indeed be no expectoration to examine, and, if there is, it often does not contain bacilli, owing to the fact that the tubercular nodules are in process of formation, and not of softening and discharge. The examination should, however, always be made and be frequently repeated.

Miliary tuberculosis limited to the lungs does not, like acute general tuberculosis, simulate *typhoid fever*, as extreme rapidity of breathing and cyanosis and marked emaciation are not present at an early stage of typhoid fever, when the difficulty of diagnosis is greatest.

A distinguishing feature between acute tuberculosis and typhoid fever is that in the former disease the course of the temperature is less regular than in the latter, and it may be of the inverse type; also in acute tuberculosis the temperature may fall to the normal or below it in the morning. This applies to cases of general acute tuberculosis, but is not observed when miliary tuberculosis of the lungs supervenes upon chronic pulmonary tuberculosis. The blood serum test may be, if conducted by a competent observer, a reliable method of distinguishing between typhoid fever and tuberculosis. A positive reaction is pathognomonic of typhoid, but a negative reaction, especially in the first few days of the disease, does not exclude that diagnosis. The urine should also be examined to see whether Ehrlich's reaction can be obtained.

The previous existence of pulmonary symptoms or of tubercular disease of glands, bone, or joints, or of the epididymis or testes, often suggests the true nature of the case, and the family history of the patient may also aid the diagnosis.

If the cerebral meninges or the choroid become involved in the spread of the disease, any doubt which may still remain as to the diagnosis usually disappears.

In *acute bronchitis of the smaller tubes* the general aspect of the case is not unlike that of miliary tuberculosis, and if catarrhal pneumonia and acute dilatation of the bronchi are also present, the physical signs may suggest that softening of a tubercular deposit is in progress throughout the lungs.

But in the form of disease under consideration the miliary tubercles do not as a rule break down, and this fact alone should prevent error. There may, however, be a limited degree of softening at one apex.

Prognosis.—It has been suggested that miliary tuberculosis of the lungs may undergo arrest, but if this ever happens after extensive infiltration has occurred, it must be an event of extremely rare occurrence. In the course of a considerable pathological experience the writer has never observed appearances indicating such a condition, and the recorded cases are far from conclusive. The disease generally ends fatally in from ten days to about three weeks, but life may be prolonged for three months, or even for a longer period.

Treatment.—The indications are to maintain the strength of the patient by rest and the administration of an abundant or of what may even apppear to be an excessive quantity of food, which should chiefly consist of milk and eggs, and to relieve those symptoms which cause the most suffering. We may be allowed to hope that, as a result of bacteriological research, a remedy will be found which will arrest the disease at the outset; unfortunately, at present we know of none.

CHAPTER XXXI

PULMONARY TUBERCULOSIS

(continued)

CASEOUS TUBERCULOSIS OF THE LUNGS

	PAGE
Etiology	336
Morbid anatomy	337
Symptoms	339
Physical signs	339
Course	340
Diagnosis	340
Prognosis	341
Treatment	341

In this variety of the disease the lesions may be widely scattered throughout the lung, the disseminated or *broncho-pneumonic* type; or involve only a single lobe, the *lobar* type.

Etiology.—The *disseminated* form is more common than the lobar, but the two together constitute only a very small percentage of the total cases of tubercular disease of the lungs. The proportion has been variously estimated at from 2·6 to 19 per cent. Of the cases analysed by the writer, which, however, did not include any children, the percentage was 2·4. Cases of the lobar variety are extremely rare.

The disseminated form is most common in children and young adults, especially females, with a marked hereditary predisposition to tuberculosis; but it may occur in persons who had previously enjoyed good health, but had recently suffered from overwork or anxiety. Children are often infected during convalescence from an acute disease, such as measles or whooping cough. The lobar form is more often met with in adults than children.

The disease usually occurs as a primary pulmonary affection, but it may be secondary to a chronic tuberculosis of the lungs. We have never observed it as a sequel of the complete arrest of tuberculosis, but its occurrence under such circumstances is described. It is, however, certainly not nearly so commonly met with then as miliary infiltration.

Morbid anatomy.—In the *disseminated* variety the process, which is from the outset not only tubercular but also acutely inflammatory in character, commences in the bronchioles; thence it quickly spreads to the alveoli, thus presenting a distinctly bronchopneumonic type. It was possible, previous to the discovery of the tubercle bacillus, to hold with Niemeyer that the inflammatory lesions present in the lungs in 'phthisis' are primary and of the first importance, and that they may lead to destruction of the organs without the aid of any specific virus; but this view is no longer tenable. A cellular exudation, consisting in part of epithelial cells and in part of more distinctly tubercular products, fills both bronchi and alveoli, and undergoes a rapid caseous necrosis. In the early stage it appears to the naked eye as greyish-red foci; these enlarge, and coalesce with neighbouring areas to form masses of varying size. The obliteration of the vessels as the exudation increases gives a dull white colour to the consolidated areas, and when caseation is complete they present the typical yellow cheesy appearance. The lobular outline is for some time preserved, and even when the lesion is diffused over a large surface, air-containing tissue, usually much injected, occupies the spaces between neighbouring masses, or the intervening lung may be in a condition resembling red hepatisation.

The caseous masses present a finely granular surface, and are either dry and friable or of a moderately firm consistence. Lesions of this nature may be irregularly scattered throughout one or both lungs almost from apex to base. Smaller nodules in the form of numerous soft white opaque, grey or caseous tubercles are also present. Softening may be in progress at numerous points, or extensive cavities may have formed; both changes are usually most advanced at the apices, but single small cavities may be found both there and in the lower lobes, and may present a fairly well-defined outline after the complete softening or the discharge of their caseous contents. In some less acute cases the consolidated areas present in part a grey or reddish-grey or gelatinous appearance.

When excavation has occurred, the bronchi may be dilated and open directly into the cavities. Recent pleural adhesions may be present, or there may be evidence of acute inflammation of the pleura, or the membrane may be studded with miliary tubercles.

The *microscopical appearances* present in such cases are various. The alveoli are seen in places to be filled with epithelial cells and with small-celled growth from the walls. The latter and the septa are thickened from a similar infiltration, which causes a narrowing of the lumen of the alveolus. The capillaries are obliterated at an early stage of the process. Where the process is more advanced all traces of cell structure have disappeared, and only an amorphous or finely fibrillated granular caseous débris is present. A small-celled growth is abundant around the bronchioles and in their walls, and also around the vessels. Elsewhere a similar infiltration is found, either in agglomerated masses or diffused throughout a considerable tract of tissue. The smaller bronchi may be filled with dried secretion. Giant cells may be found, and tubercle bacilli are usually present in considerable numbers.

The effect of a widespread infiltration of tubercle accompanied by inflammatory changes in the intervening parts of the lung, when the process is somewhat less acute than that just described, is to produce an area of consolidation of a more or less uniform grey colour. It is at first soft and presents a slightly granular surface and breaks down easily on pressure, but at a later period it becomes firmer in texture, and has a glistening or gelatinous appearance on section.

The stage of grey infiltration is probably preceded by one to which the term 'red infiltration' has been applied, in which the lung still contains blood and is more granular and opaque on section and of softer texture, as in some cases tissue presenting this appearance may be seen to merge into an area of grey infiltration.

The stage of grey infiltration may be followed by caseation, which usually commences at numerous well-defined foci in an area of infiltration, the size of the caseous nodules varying from that of a pin's head or a pea to that of a walnut. Softening of the infiltrated areas very rarely occurs without the previous appearance of caseation.

In the *lobar* variety of the disease the appearances presented post mortem are far from uniform, but they vary less widely than in any other type of the disease. The lesion may be limited to a single lobe, or more rarely almost the whole of one lung may be involved. The consolidation is seen to be as complete as in a case of pneumonia. On section the affected area is caseous throughout, yellow or yellowish white or greyish white in colour, dry and airless. The surface is smooth and cheese-like or finely granular. No tubercular foci are visible as a rule, the change being uniform over the affected part. In other cases softening may be in progress at the apex, for the upper lobes are most often affected, and a cavity may have formed. The margin is ragged, and there are no signs of limitation of the destructive process, such as are afforded by the presence of a distinct wall; the contents of the cavity consist of puriform débris. In other cases the section presents a greyish-white appearance, is less firm in consistence, and more granular on section. The evidence of the tubercular nature of the change is then more obvious, aggregated masses of granulations in part constituting the consolidated areas. The appearances as a whole are then less uniform, caseous areas being in places bordered by consolidated tissue which is hyperæmic. If the change occurs in an emphysematous lung, the surface of the section may present a worm-eaten appearance.

The pleura over the affected areas is generally inflamed, and may be covered with pale adherent exudation. Necrosis of the pleura followed by perforation and pneumothorax is especially likely to occur in this variety of the disease.

The bronchi [illegible] re enlarged, and often caseous.

Secondary tubercular lesions may be present in the larynx, trachea, and intestines.

In some cases the true nature of the changes found in the lungs is not obvious until the whole of the organs have been examined, when typical tubercular lesions are discovered elsewhere.

Symptoms.—The onset may be sudden or more or less insidious. In the former event there may be repeated rigors, in the latter languor, anorexia, pains in the limbs, cough, slight pyrexia, and progressive loss of strength may precede its complete development. In some cases hæmoptysis is the first symptom noticed. In nearly all cases after a short time the general symptoms become well marked, there is high fever, rapid emaciation, a hectic flush on the cheeks, burning heat of the skin, night sweats, chills, and evidence that disease is rapidly extending throughout the lungs. The expectoration is at first mucoid and may be blood-stained, but soon becomes mucopurulent or puriform, and has been observed in some cases to present a greenish tint. In a very acute case recently under the observation of the writer, it was of a bright lemon-yellow colour. Tubercle bacilli are almost invariably present, and in considerable numbers. The pulse is extremely rapid, and respiration is as a rule markedly quickened, the ratio being very often three or even four to one. The character of the pyrexia has already been described (*vide* p. 327).

In the *lobar* form of the disease the onset of the acute illness may have been preceded by a period of impaired health, and the clinical history of the patient may indicate a tendency to pulmonary disease; but in many cases the previous health has been good, and there may be no evidence of tubercular disease in the family. The patient, possibly after exposure to a chill, is suddenly seized with a severe rigor followed by pain in the side, the attack in this, as in other respects, resembling one of pneumonia; there is high fever, cough, urgent dyspnœa, and rusty expectoration, and for a week or ten days the true nature of the disease is likely to remain unsuspected.

Physical signs.—These usually appear first at the apices, loss of resonance on percussion being accompanied by crepitation. In the case just referred to, in which the sputa were of a lemon tint, the signs of consolidation were first observed just external to the left nipple, and from there the disease spread rapidly over the lower lobe and to the opposite lung. Very often the first signs are diffuse râles over both lungs, at first bubbling in character, but rapidly becoming of a crackling or metallic quality.

The breath sounds may be bronchial, but typical tubular breathing is not often heard. This is due, no doubt, to the bronchi of the consolidated areas being filled with exudation. Bronchophony is commonly present. The formation of cavities is attended by the usual signs. A pleural friction sound is often present, and may be followed by signs indicating that effusion has occurred.

In the *lobar* variety the physical signs are those of pneumonia —viz. dulness on percussion, with crepitation and tubular breath

Nothing, indeed, may be wanting to justify a diagnosis of pneumonia, and cases are almost invariably for a time regarded as of that nature, no doubt arising until the period has elapsed during which a crisis generally occurs.

The absence of the crisis, a more rapid pulse, sweating, and an unusual degree of emaciation then suggest an examination of the sputa for tubercle bacilli, and their discovery reveals the real nature of the disease. Bacilli may, however, be absent for a time, and may only be found after repeated examination, and when the sputa have assumed a mucopurulent character.

Course.—The disease is characterised by a rapid course, a fatal termination having been observed in so short a time as a fortnight. This is, however, quite exceptional, the ordinary duration of an acute case of the broncho-pneumonic type being from two to three months. A remission of the acute symptoms may, however, occur after a variable period, and the case may assume a subacute or chronic character. This is occasionally observed to occur in children after an acute illness lasting from a fortnight to three weeks.

Death may be due to rapidly increasing prostration and emaciation, or, as is frequently the case, to the occurrence of pneumothorax, the pleura over the central portion of a caseous patch ulcerating before adhesions have had time to form.

An attack of hæmoptysis may also be the immediate cause of death, but hæmorrhage is not common in this variety of the disease.

Gangrene of the lungs is met with occasionally, and quickly proves fatal.

The course of the disease in cases of the lobar type is often still more rapid. A fatal termination may occur in so short a time as a fortnight, while the longest duration is rarely more than three or four months. The lung may be found still solid post mortem, or softening may have occurred, usually at the apex, where cavities are in course of formation.

Diagnosis.—In all cases of pulmonary disease, no matter how innocent their seeming character, the sputa should be examined for tubercle bacilli. If this rule be observed, a correct diagnosis will generally be made in cases of caseous tuberculosis. The discovery of elastic tissue in the sputa is a sign that the lung is breaking down. In children, however, these tests may not be applicable, because the expectoration is usually swallowed, and in such patients a diagnosis of broncho-pneumonia is not uncommonly only disproved by a post-mortem examination. The fact, however, that diffuse broncho-pneumonia attended by acute dilatation of the bronchi may give rise to physical signs for a time indistinguishable from those of disseminated caseous tuberculosis suggests caution in giving an unfavourable opinion. When there are unmistakable signs that the lung is breaking down, and tubercle bacilli are present in the sputum, the real nature of the case becomes clear, and in doubtful cases it is well to wait for their appearance before committing oneself.

It will be apparent from the description of the *lobar* form of the affection, that pneumonia is the disease for which it is most likely to be mistaken, and in the early stages the error is not only pardonable, it may even be said to be necessary, and, provided the true character of the disease is recognised as soon as the evidence is clear, no harm is done. These cases are of very rare occurrence, and any one who feared that every case of ordinary pneumonia he observed might prove to be of this nature would by his unusual alertness probably do more harm than good.

Prognosis.—It follows from the description of the disease that this is in all cases unfavourable. It is, however, not quite so grave in the broncho-pneumonic as in the lobar form. In the less acute cases it may fairly be hoped that the disease may pass into a subacute or chronic stage.

Treatment.—It is of the first importance to maintain the strength of the patient, as the only hope of an abatement in the severity of the symptoms lies in the recovery of a resisting power which may have been lost through temporary causes. There may be some chance of this if appetite is retained, as it sometimes is, but very little otherwise. Complete rest in bed is essential, the air of the room being kept as fresh as possible. Not only abundance but what might be considered an excess of nourishing food should be given, chiefly in a liquid form, the only limit to the quantity being the ability of the organs of digestion to deal with it satisfactorily. No drugs should be administered which are at all likely to produce nausea or impair digestion. All lowering remedies are to be avoided, particularly those of this character so often given to diminish the pyrexia, their effects being in the writer's experience distinctly harmful. The temperature certainly falls, but no effect is produced upon the course of the disease, and when they are omitted there is an immediate return of the fever. Sponging the body with water at a temperature of 85° F. is often decidedly refreshing to the patient; it lowers the temperature and exercises a general tonic effect. It should be done in the afternoon or evening, when the fever is at its highest point. Toilet vinegar or eau-de-Cologne may be added to the water with advantage. A saline effervescing mixture containing two grains of quinine may with advantage be given every three or four hours. Food should be given at intervals of about two hours, and should consist of milk, jelly, beef tea, meat essence, and chicken broth. Stimulants, of which old brandy is probably the best, may be needed in considerable quantity.

The detailed consideration of much of this part of the subject may conveniently be deferred to the chapter on Chronic Pulmonary Tuberculosis (*vide* p. 404, Treatment of Pyrexia).

J. K. F.

CHAPTER XXXII

PULMONARY TUBERCULOSIS

(continued)

FIBROID TUBERCULOSIS OF THE LUNGS

PAGE
Etiology 342
Morbid anatomy 342
Symptoms and course 344
Physical signs 344
Diagnosis 345
Prognosis 345
Treatment 345

Etiology.—The subjects of this form of the disease are often of good muscular development, and possess a resisting power which is greater than is met with in any other. This is shown by the tendency of the disease to undergo arrest, its slow course, the fibrous transformation of the tuberculous products, the few bacilli present in the lesions and in the sputa, and by the rare occurrence of infiltration generalised throughout the body.

Men are more often affected than women, and the period of onset is later than in the chronic variety.

There is often no family predisposition to tuberculosis, but in one of the most marked examples known to the writer several brothers had suffered from tuberculous disease.

The tendency to fibrosis is increased in those individuals whose lungs have been rendered vulnerable by inhaling irritating particles of dust; the affection is therefore common amongst workers in various trades—*e.g.* knife-grinders.

Morbid anatomy.—The apex of the lung, as in other forms, is the site of the primary lesion. The typical fibroid lesions consist (*a*) of isolated granulations which are indurated and deeply pigmented, (*b*) of groups of such granulations having a racemose arrangement, and (*c*) of areas of induration either rounded or irregular in outline, at the margins of which fibrous granulations are present. The central part of such areas is of a coal black colour, of a fibroid or cartilaginous density, and shows no trace of the presence of granulations, the section being smooth and homogeneous.

The disease begins, as in the caseous form, in the finer bronchi, but its extension is more toward the peribronchial tissue than toward the alveoli. The primary lesion consists of tubercles which present the structure typical of such bodies, but instead of undergoing caseation and softening they retain their rounded outline and become transformed into firm, fibrous, deeply pigmented bodies, which look and feel like shot embedded in the tissue of the lung. On microscopical examination the bronchus is seen to be filled with round and epithelial cells and the bronchial wall to be thickened from small-celled infiltration. Tubercles are present both in the wall of the bronchus and in the peribronchial tissue. The inflammatory changes in the alveoli are much less marked than in the caseous form.

The larger areas of induration are in some cases formed by the coalescence of granulations presenting a racemose arrangement, which extend by the formation of tubercle at their margins, and, as they increase in size, undergo transformation into pigmented fibrous tissue in the centre. To these racemose masses the name of 'Carswell's grapes' has been given.

Fibrous thickening of the interlobular connective tissue is generally present, and may lead to the formation of bands which either unite the more densely indurated areas, or are so thickly set as to form tracts of fibroid tissue having a netlike structure. Racemose tubercles may also be present at the margins of these areas.

In some cases areas of grey infiltration may be seen undergoing a fibroid transformation.

In lungs presenting these appearances small caseous or calcareous masses may be found, but they are by no means constant. They are in some cases due to caseous change in tubercles, in others to the inspissation of the contents of the bronchi. A single cavity or several of small size may be found at the apices or elsewhere; they are usually smooth-walled and surrounded by indurated tissue. Their presence is by no means constant, and possibly indicates that the primary disease was of the ordinary chronic type, the fibroid change taking place with a return of resisting power in the individual. Except in cases of this kind the bronchi are rarely found dilated.

The contraction of the fibrous areas is generally accompanied by emphysema of the surrounding tissue. If the lesion is near the surface there may be adhesions, with scarring and puckering of the pleura, and large bullæ may project from the surface of the lung. But the extreme thickening of the pleura which is so commonly found in cases of chronic tuberculosis is rarely if ever present in these cases.

A few bacilli may be found in the more recent lesions, but they are absent from the dense pigmented areas, and can rarely be discovered in arrested fibrous lesions at the apices.

Symptoms and course.—The onset is never acute, and in some cases, after a moderate area of the apex of one lung has become involved, the process may undergo arrest, and there may be an entire absence of all symptoms from that date. If, indeed, one may trust to the clinical history given by many patients found after death to have arrested lesions at one or both apices, the symptoms could not at the time of the formation of the deposits have been very marked, as there is rarely any reference to a previous pulmonary affection. Cough, with loss of weight and strength, and probably a moderate degree of pyrexia, are the only symptoms likely to have been present.

As the fibrous area increases in size and more of the surrounding lung becomes emphysematous, the patient suffers from dyspnœa, becomes more subject to catarrh and wheezing, and may have attacks of 'bronchial asthma.' In some cases, however, there is not the slightest tendency to catarrhal affections. In delicate-looking individuals, the subjects of emphysema and liable to attacks of bronchial asthma, the existence of an arrested or slowly extending fibroid tuberculosis of the lungs should always be suspected. Hæmoptysis in the early stages is very common; it may be profuse and frequently recur, but recovery quickly follows each attack, and there may be no physical signs indicating that the disease in the lungs has extended.

The typical apyrexial course of the disease has been already described. It may be interrupted by short periods during which fever is present; but the rise of temperature is, as a rule, only moderate, and there may indeed be complete absence of fever with an extension of the disease. The temperature is often subnormal in the morning.

The cough is rarely very severe, except when associated with catarrh and emphysema, when it chiefly occurs in the morning. It has not the paroxysmal character and the tendency to produce vomiting, which are common in chronic tuberculosis with marked retraction of the chest, thickening of the pleura, and excavation of the lung ('fibroid phthisis'). The disease rarely extends to the larynx, and there is an absence of night sweats and diarrhœa. There may be little or no expectoration, and the sputa may be free from bacilli for long periods. In a case under the care of the writer bacilli were only found after repeated examinations extending over two months, and on subsequent re-admission to hospital after an interval of nearly a year, during which the disease had apparently remained quiescent, none were found.

In advanced cases the extensive area of the lungs involved is often in striking contrast with the apparent well-being of the patient, the appetite being good and emaciation but little marked.

If there is a considerable degree of emphysema, epigastric pulsation and other signs of that condition will be present, and the general aspect of the patient and the alterations in the shape of the thorax will be such as accompany that disease.

Physical signs.—Some retraction of the supra and infraclavicular regions of one side with impaired expansion may be observed, but if emphysema has developed around a contracted lesion retraction may not be noticeable. The condition of the vocal fremitus and resonance will depend upon the extent of the induration and its nearness to the surface of the lung, and also upon the presence or absence of emphysema of the intervening pulmonary tissue. There may be slightly defective resonance on percussion, or the note may indicate the presence of emphysema. The breath sounds are usually weak and expiration is prolonged. Fine crackling râles, sometimes only audible with cough and accompanied by signs of catarrh, are the common adventitious sounds. The râles may be widely distributed, and, as in the chronic form of the disease, the lesions tend to spread from the apex of the upper to that of the lower lobe, and thence towards the base. The presence of a cavity, if it be of sufficient size, will be indicated by the ordinary signs; such cavities are usually dry.

Diagnosis.—The existence of the tubercular lesion may be masked by the signs of emphysema and bronchitis, but a careful examination of the apices and particularly of the supra-spinous fossæ will generally lead to its recognition.

The fact also that the signs are more marked over the upper lobes than at the bases should always excite suspicion.

Repeated examination of the sputa should be made for tubercle bacilli; if this is done, a few will generally be found at some period of the disease.

It is often, however, impossible to be certain of the presence of an arrested fibroid lesion occupying only a small area and surrounded by emphysematous lung.

Prognosis.—The recognition of this type of the disease is of great importance from the point of view of prognosis, owing to the fact that fibroid tuberculosis tends more than any other variety to undergo arrest, or failing that to run a very prolonged course.

Complete and permanent arrest, however, rarely occurs when extensive areas of the lung have undergone induration; the progress is slow and may be interrupted by periods of quiescence, but, as a rule, the patient dies of some pulmonary affection.

The **treatment** of cases of this type does not differ from that suitable in the chronic form of the disease (*vide* p. 385 *seq.*).

J. K. F.

CHAPTER XXXIII

PULMONARY TUBERCULOSIS

(continued)

CHRONIC OR FIBRO-CASEOUS PULMONARY TUBERCULOSIS

PAGE
Morbid anatomy 346
Classification of cavities 348
Site and progress of the lesions 350
    Upper lobe 350
    Middle „ 352
    Lower „ 352
  Crossed lesions 355
  Basic „ 356
Lesions associated with pulmonary tuberculosis 358

Morbid anatomy.—In this form of the disease the appearances present an almost infinite variety, but cavities are rarely absent.

These, with the primary lesions and the secondary changes which they subsequently undergo, together constitute its morbid anatomy, the presence of miliary granulations and areas of caseation indicating that the chronic course of the disease has been marked by periods during which the morbid process has been active. Such a period of activity often immediately precedes the death of the patient, and during its continuance miliary tubercles in great numbers may be formed in the parts of the lungs hitherto unaffected.

The chronic nature of the process is revealed by induration of the granulations, in extensive tracts of consolidation, and in the walls of the cavities. The more prolonged the duration of the case, the greater is the amount of fibroid change produced.

The miliary, caseous, and fibroid lesions have already been described in considering the varieties of the disease in which they severally predominate. Their mode of combination, and the secondary changes which they undergo, and also the lesions which are more or less special to this form of the disease, will now be described.

Granulations and nodules.—Miliary granulations tend as a result of age to undergo fibrosis and pigmentation, and caseous nodules to become firm, dry, and granular. Caseous nodules which have coalesced are often seen to be surrounded by a zone of dense pigmented tissue, from which fibrous striæ penetrate towards the centre. Sometimes the caseous areas are limited by tracts of semi-transparent indurated lung or a well-defined fibrous capsule.

Caseous nodules may, from the deposit of lime salts, become calcareous, and in such a condition are often found encapsuled. The destruction of the capsule owing to recrudescence of the disease in the neighbourhood may lead to the escape of such masses into a bronchus, and their appearance in the expectoration.

The pigmented racemose nodules found in the fibroid variety of the disease are often present, and from their margins fibrous bands may be seen extending into the tissue around.

Infiltrations and indurations.—When a large number of tubercles are present in a given area they may, as in cases of miliary tuberculosis, remain discrete, and be separated by lung tissue which is in a more or less normal condition, but in the majority of cases the tubercular foci are not thus distinctly circumscribed, and changes are produced in the walls of neighbouring alveoli and bronchioles. These become thickened and infiltrated with small cells, and the alveoli filled with epithelial cells, leucocytes, and sero-fibrinous exudation. These changes as a whole result in the production of an area of consolidation of a grey colour, firm consistence, and, if of comparatively recent formation, granular appearance on section. Caseous foci are commonly present in such an area of infiltration: of these some may be undergoing softening, whilst others are firm, dry, and granular.

The tracts of grey infiltration become indurated with age, and may acquire an almost cartilaginous density. They are then smooth and glistening on section, but in the less chronic forms they may still present a slightly granular appearance. Pigmentation is a characteristic change in old lesions of whatever character.

Areas of fibrosis, usually deeply pigmented, may occupy extensive tracts of the lung primarily affected; they are formed by the coalescence of indurated granulations and nodules, or by the increase in thickness of fibrous bands extending from the margins of conglomerate masses of tubercles which have undergone the fibrous change.

The fibrous cicatricial tissue contracts and becomes puckered, and is often traversed by dilated bronchi of a fusiform shape which terminate in cavities.

Pneumonia.—How far the ordinary processes of inflammation, particularly those of the catarrhal form, take part in producing the morbid appearances found in the lungs in 'phthisis' is a question which has given rise to prolonged controversy, but it is one which probably excites less interest at the present time than formerly.

It is true that from the histological appearances it may be

The writer drew attention some years ago[1] to the fact that 'tubercular disease in its onward progress through the lungs in the majority of cases follows a distinct route, from which it is only turned aside by the introduction of some disturbing factor,' and subsequent experience has confirmed the correctness of the views then expressed.

This 'line of march' of the disease is characteristic of cases of the chronic and fibroid varieties in which the progress of the disease is slow, but is less frequently observed in the caseous variety, which may be marked by rapid extension from lobe to lobe, and from one lung to another. Time is necessary for the temporary localisation of the disease which such a mode of progress requires, but there is rarely any inversion of the natural order; the disease spreads in each lobe from above downwards, hardly ever from below upwards.

The site and progress of the lesions.—The apex of the lung is the primary site of the disease in the large majority of cases. Of the many theories which have been put forward in explanation of this fact, that which attributes it to the lesser functional activity of the part appears on the whole to be most worthy of acceptance.

In quiet automatic breathing the apices of the lungs are imperfectly expanded. This is true even of the female, in whom the pectoral regions, rather than the apex proper, are functionally most active. Impairment of function probably leads to a defective resisting power, and thus the tubercle bacillus is able to effect a lodgment at the apices, although in other areas of the lungs the normal forces of the body are sufficient to insure its destruction.

Lebert attributes the great proclivity of the apex to the disease to the effect of posture. He does not explain the influence of the erect position, but is probably correct in attributing to the supine posture an influence in promoting the extension of the lesions to the posterior aspect of the lungs.

Rindfleisch explains the apical site of the primary lesion upon the hypothesis that the bronchial secretions there are thicker than in other parts, owing to the relative dryness of the apices from the subsidence of the blood, the effect of gravity.

The supposed larger size of the alveoli at the apex of the lung, of which there is no proof, and the greater exposure of the upper parts of the lungs to the influence of cold air, are other explanations of the common site of the primary lesion which do not need detailed discussion.

Sites of lesions in the upper lobes.—The extreme apex of the lung is not often the site of the primary lesion; this usually occupies the situation marked on the accompanying diagram (fig. 96). It is situated from an inch to an inch and a half below the summit of the lung, and rather nearer to its posterior and external borders. Lesions in this situation tend to spread in the first instance backwards, possibly from inhalation of the virus whilst the patient is lying down. This line of extension explains the fact that an examination of the supra-spinous fossa will often give certain evidence of the presence of disease when the physical signs observed beneath the clavicles are of doubtful import; proving that the changes, as is generally the case, are more advanced behind than towards the anterior aspect.

---

[1] *Localisation of the Lesions of Phthisis*, p. 11 (1888).

From this primary focus, which in front corresponds either to the supra-clavicular fossa or to a spot immediately below the centre of the clavicle, the lesions often spread downwards along the anterior aspect of the upper lobe, about three-fourths of an inch within its margin, frequently occurring in scattered nodules, separated perhaps by an inch or more of healthy tissue. It is not unusual to find in these scattered nodules the only evidence of disease on the anterior aspect of the lung, when posteriorly excavation has advanced to such a degree that but little more than the pleura remains.

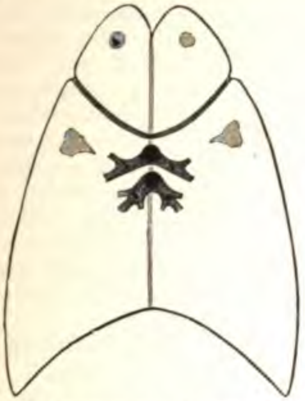


FIG. 96.—DIAGRAM OF A VERTICAL MEDIAN SECTION OF THE LUNG, SHOWING ONE OF THE SITES OF THE PRIMARY INFILTRATION IN TUBERCULOSIS; ALSO THE SITE OF EARLY INFILTRATION OF THE LOWER LOBE

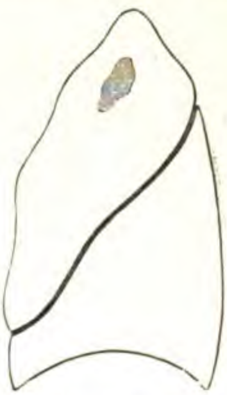


FIG. 97.—DIAGRAM OF THE LEFT LUNG, VIEWED FROM ITS OUTER BORDER, SHOWING A LESS USUAL SITE OF PRIMARY DISEASE OF THE APEX

A second and less usual site of the primary affection of the apex is seen in fig. 97. This corresponds on the chest wall with the first and second interspaces below the outer third of the clavicle. The lines of extension are usually downwards, so that after a time an oval area of lung is involved, occupying the outer part of the upper lobe. It is possibly true that the spread of the disease is more rapid when the primary lesion occupies this site. The lesions in the advanced stages—indurations and cavities—are formed by the coalescence and extension of these primary foci. The scattered nodules of consolidation on the anterior surface of the lung often unite and break down, forming a long sinuous cavity which may extend almost to the lower margin of the upper lobe anteriorly; posteriorly, where the signs of excavation are usually most distinct, the further progress of the disease is generally arrested at the pleural reflexion in the interlobar septum. This was found to be destroyed, and the cavities in the upper and lower lobes united, in only five out of one hundred and fifty-two consecutive cases of the disease examined post mortem by Dr. Ewart.[1]

Site of lesions of the middle lobe.—The middle lobe of the right lung (which is believed by Aeby, from the arrangement of the bronchi, to be the analogue of the upper lobe of the left), is rarely the site of a primary tuberculous lesion. It is almost invariably affected after the upper lobe of the same side and usually at a rather late period of the disease, whilst not uncommonly it escapes altogether. The lesions most commonly found there are coarsely granular nodules, often of large size, which are either undergoing caseation, or already present an area of softening in the centre.

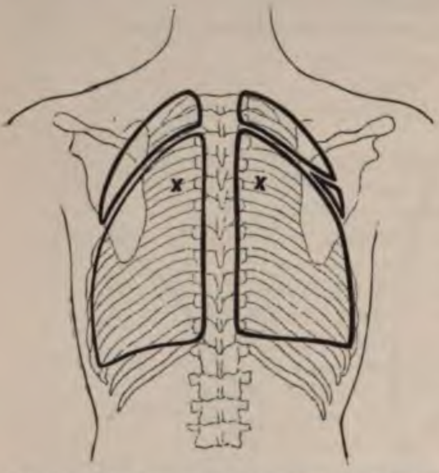


FIG. 98.—SHOWING THE RELATIONS OF THE LOBES TO THE WALL OF THE CHEST POSTERIORLY

The X marks the usual site of early infiltration of the lower lobes.

Extension to the lower lobe. — The lower lobe of the lung primarily diseased is usually affected at a very early period of the disease, often long before any extensive infiltration or destruction of the upper lobe has taken place, and as a rule before the apex of the opposite lung is attacked.

The site of the secondary infiltration of the lower lobe is indicated in figs. 98 and 101. It is situated about an inch to an inch and a half below its highest point, although it may sometimes be nearer to and even at the apex of the lobe. This situation nearly corresponds on the chest wall to a spot opposite the fifth dorsal spine, midway between the border of the scapula and the spinous processes of the vertebræ.

The infiltration of the lower lobe at this site in the early stage of pulmonary tuberculosis of the chronic and fibroid varieties is one of the most constant features in the pathological anatomy of the disease, and its recognition is a point of the greatest clinical importance, as the existence of a lesion in the lower lobe at this spot, coincident with physical signs at the apex, even though the latter are in themselves of doubtful import, is in the writer's experience almost positive proof of the presence of tubercular disease of the lung. It may be of great service in cases in which tubercle bacilli are absent from the sputa.

---

[1] *Goulstonian Lectures*, 1882.

It would therefore appear that the upper and posterior part of the lower lobe is a spot only second in point of vulnerability to the apex itself. It is very rare, except in cases of 'crossed lesions' (*vide* p. 355), to meet with a case, either during life or on the post-mortem table, in which this area is affected whilst the apex of the lung is free from disease.

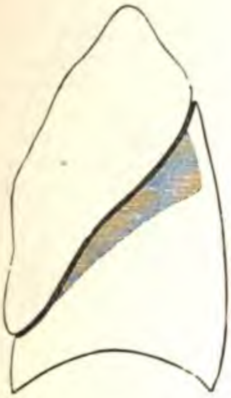


FIG. 99.—SHOWING THE LINE OF EXTENSION OF A LESION OF THE LOWER LOBE ALONG THE INTERLOBAR SEPTUM

Lateral aspect of the lung.

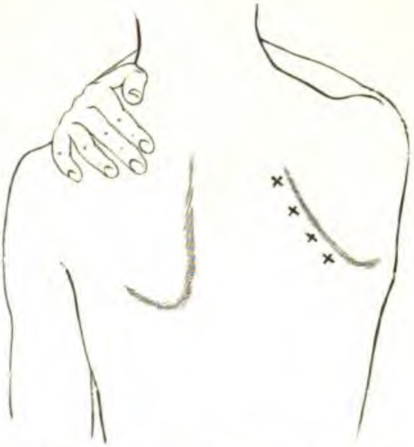


FIG. 100.—SHOWING THE POSITION OF THE ARM WHEN THE VERTEBRAL BORDER OF THE SCAPULA INDICATES (ROUGHLY) THE USUAL LINE OF EXTENSION OF LESIONS ALONG THE INTERLOBAR SEPTUM

Cases in which active lesions are present at both apices, no other portion of either lung being affected, are rare, but both apices may be the seat of old disease when the process has been arrested before the lower lobe on either side has become affected.

The early stage of the disease at which the lower lobe is implicated is well illustrated in lungs presenting old apex lesions which have undergone arrest. The area of disease may not be larger than a cherry or an olive, but if the process was tuberculous, a nodule will generally be found in the lower lobe. This lesion, once established, tends to spread backwards towards the posterior surface of the lung, and laterally along the line of the interlobar septum (fig. 99), forming a wedge-shaped area of infiltration. It follows from this that even in the early stages of the disease, in order to ascertain the extent of lung affected, the lower lobe must be examined not only opposite the fifth dorsal spine, but also along the line of the interlobar septum. This line is roughly marked by the vertebral border of the scapula when, with the hand upon the spine of the opposite scapula, the elbow is raised above the level of the shoulder (fig. 100). The mode of **extension towards the base** of the lung is not usually by an advancing line of consolidation, but by scattered nodules of infiltration often arranged in a racemose manner (fig. 101). Even at the termination of a chronic case some healthy or at least uninfiltrated tissue will generally be found at the base, even of the lung primarily affected. This freedom of the bases from tubercular lesions is another very marked feature in the pathological anatomy of the disease.

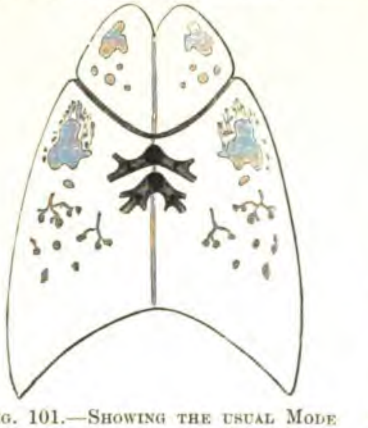


Fig. 101.—Showing the usual Mode of Extension of the Disease towards the Base of the Lung

Section from the posterior border towards the root; opened out

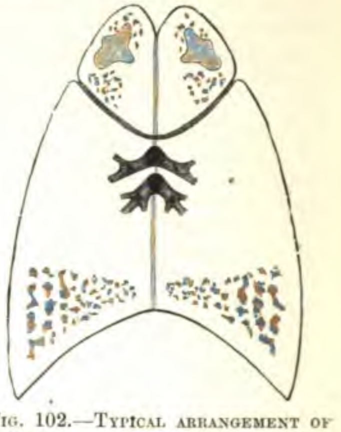


Fig. 102.—Typical arrangement of Lesions in a Case of Apical Tuberculosis with Non-tuberculous Disease of the Lower Lobe

In estimating the probability of any basic lesion being tuberculous in origin, it is important to observe whether the physical signs of disease in the lower lobe are continuous from its apex to its base; if so, the lesion is probably tuberculous. If the base is affected, but the apex of the lower lobe free from disease (*vide* fig. 102), the basic lesion is either non-tuberculous—*e.g.* due to œdema and collapse, followed by bronchiectasis, catarrhal pneumonia, or pleurisy, &c.—or, if tuberculous, the resisting power of the base has been diminished by some previous affection—for example, an attack of pleurisy followed by partial collapse; but the presumption is strongly in favour of a non-tuberculous lesion.

Mode of extension to the opposite upper lobe.—The upper lobe of the lung not primarily affected is often attacked at an early period, but not, as a rule, until after the lower lobe of the lung first affected.

The lesions may be found in either of the common situations indicated in figs. 96 and 97, and are therefore symmetrical in site but in different stages on the two sides.

There is, however, a third site for the secondary infection of the opposite upper lobe, which is figured in the accompanying diagram (fig. 103). Its site is close to the interlobar septum, about midway between its upper and lower extremities, and corresponds on the chest wall to the upper part of the axilla. There small areas of consolidation form and coalesce, but rarely break down into a cavity of any considerable size. Once established, this lesion tends to spread in nearly all directions, and may occupy a considerable area of the lung whilst the apex is free from disease.

Infiltration at this site, when present, is generally secondary to that of the lower lobe of the lung first affected.

It follows from this fact that, in addition to the apex of the lung, the part corresponding to the axilla must be carefully examined before (say) the left lung can be pronounced to be free from disease, when the right is primarily affected.

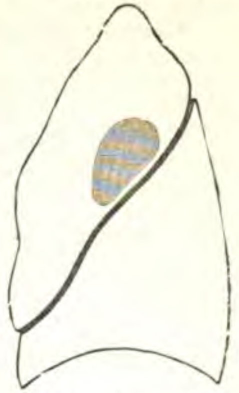
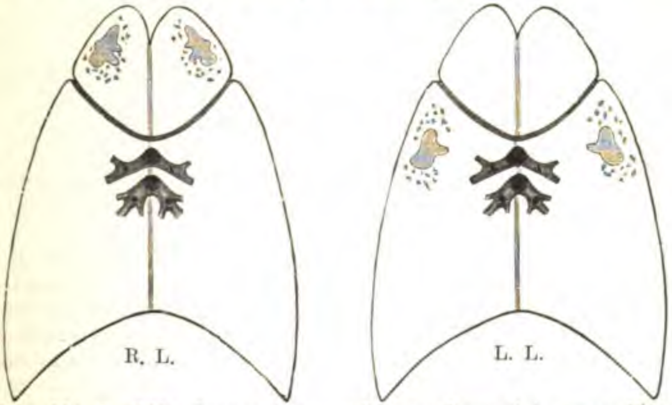


FIG. 103.—LATERAL VIEW OF LUNG SHOWING AN OCCASIONAL SITE OF LESION IN THE LUNG NOT PRIMARILY AFFECTED

Extension to the lower lobe.—The distribution of the lesions in the lower lobe of the lung secondarily diseased is usually similar to that on the opposite side, and presents no peculiarities.



FIGS. 104 AND 105.—ILLUSTRATING A 'CROSSED LESION,' *i.e.* FROM RIGHT APEX TO LEFT LOWER LOBE

Sections from the posterior border towards the root of the lung

Crossed lesions of the lower lobe.—Occasionally the lower lobe of the lung primarily affected escapes infiltration, and the disease crosses over to the opposite lower lobe (*vide* figs. 104, 105). In all cases, therefore, it is necessary to examine both lower lobes before deciding that the disease is limited to one apex.

Exceptional arrangement of lesions.—When the usual sites of infiltration in the upper and lower lobes are already occupied by arrested lesions, and a second tubercular infection of the lung occurs at some later and perhaps distant date, the more recent lesion in the upper lobe usually occupies a position close to the interlobar septum, whilst that in the lower lobe is situated along the posterior border and extends almost to the base (fig. 106).

This fact is of importance, as it helps in some cases to explain the occurrence of basic disease, and will be referred to again in the description of lesions of the lower lobes occupying exceptional situations.

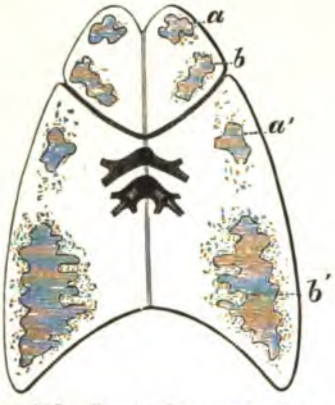


FIG. 106.—SITE OF LESIONS IN SECONDARY INFECTION

a, *a'*, arrested lesions; *b*, *b'*, recent lesions.

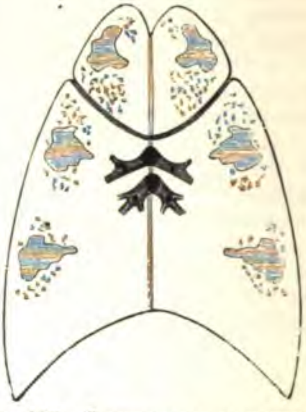


FIG. 107.—ILLUSTRATING AN UNUSUAL ARRANGEMENT OF LESIONS IN THE LOWER LOBE

Lower lobe.—The accompanying diagram (fig. 107) illustrates an unusual lesion, of which several examples have come under our notice.

A second cavity, almost equal in size to that at the apex of the lower lobe, is present about midway between that spot and the base, and close to the posterior border of the lung, the intervening area being almost free from disease and the base quite unaffected. It is, however, rare to meet with this lesion whilst the usual site of disease in the lower lobe remains unaffected.

Basic lesions.—Reference has already been made to the comparative freedom of the bases of the lungs from tuberculous disease; whenever, therefore, a lesion is found in this situation, a most careful survey should be made, both of the history of the case and of the physical signs present, before coming to the conclusion that the basic disease is tuberculous.

The most common varieties of chronic disease affecting the bases of the lungs may be classified thus:

(*a*) NON-TUBERCULOUS BASIC DISEASE

1. Collapse of the lower lobe caused by pleural effusion, followed by absorption of the fluid and falling-in of the lower part of the chest on the affected side.
2. Collapse from the same cause, followed by cirrhosis of the lung and bronchiectasis.
3. Empyema opening into the lung.
4. Hepatic abscess or hydatid cyst of the liver, communicating with the lung.
5. Collapse of the lower lobe from pressure on the main bronchi by growths or enlarged and infiltrated mediastinal glands, followed by bronchiectasis.
6. Diffuse gangrene of the lower lobe resulting from a communication through the bronchi with the œsophagus, either directly or through the medium of a softened bronchial gland.
7. Chronic pneumonia and bronchiectasis following on the impaction of a foreign body in one of the bronchi of the lower lobe.
8. Unresolved and chronic pneumonia of the lower lobe.
9. Bronchiectasis secondary to catarrhal pneumonia and collapse. This lesion is rarely found except in children.

(*b*) NON-TUBERCULOUS BASIC DISEASE COMPLICATED BY SUBSEQUENT TUBERCULOSIS

Cases presenting basic lesions are occasionally met with in which the disease, originally non-tubercular, has become so at some later period, the lower lobe being infected either directly or, as is perhaps more common, subsequently to the apex of the same lung. This complication is very likely to occur where bronchiectasis forms a part of the original lesion, but may also be found in cases of chronic pneumonia and in some of the other forms of non-tuberculous basic disease enumerated above.

(*c*) BASIC TUBERCULOSIS

1. Physical signs most marked at the base, but the oldest lesions at the apex.
2. Arrested lesions at the apex and at the posterior apex of the lower lobe, more recent lesions in the upper lobe and also at the base (*vide* fig. 106).
3. Primary basic tuberculosis.

Cases illustrating several of the conditions here tabulated are described by Dr. Percy Kidd, in a paper on 'Basic Tuberculous Phthisis,'[1] from which some of the headings here used are taken.

It is unnecessary to discuss in detail the differential diagnosis of the various forms of basic disease here enumerated, as the physical signs by which they are attended are described in the appropriate chapters. It must suffice to repeat that the only positive proof of the tubercular nature of pulmonary disease is the discovery of bacilli in the sputa.

---

[1] *Lancet*, October 2, 1886.

LESIONS ASSOCIATED WITH PULMONARY TUBERCULOSIS

In the following table, compiled from the Pathological Reports of the Brompton Hospital for the years 1892, 1893, and 1894, the more important lesions found in association with pulmonary tuberculosis are stated.

As the method of tabulation adopted in the Reports is not uniform throughout, it has not been possible to give complete figures under each heading.

—	1892	1893	1894	Total cases	Per cent. of cases included
Number of Cases	208	174	149	531	—
I. Respiratory System:					
Laryngeal tuberculosis	100	84	66	250	47
Tracheal ulceration	36	32	28	96	18
Bronchial ,,	6	10	4	20	3·7
Bronchiectasis	1	—	5	—	—
Pulmonary aneurysm	15	16	8	39	7·3
Hæmoptysis (death from)	18	15	7	40	7·5
Pneumothorax	16	14	16	46	8·6
Pyo-pneumothorax	6	7	13	26	4·8
Pleural effusion	—	12	12	—	7·4
Gangrene of lung	—	3	1	—	1·2
II. Digestive System:					
Alimentary canal—					
Tubercular ulceration of pharynx	2	3	2	7	1·3
,, ,, tongue	1	3	—	—	—
,, ,, œsophagus	—	1	1	—	—
,, ,, intestines	165	131	—	—	77·4
,, ,, stomach	—	—	2	—	—
Perforation of intestine	8	3	5	16	3
III. Abdominal Organs:					
Peritonitis—					
(1) Non-tubercular	15	10	5	30	5·6
(2) Tubercular	6	9	7	22	4·1
Liver—					
Amyloid	—	14	6	—	—
Cirrhotic	8	10	7	25	4·7
Miliary tubercle	1	4	4	9	1·6
Fatty	—	54	68	—	37·7
Spleen—					
Amyloid	—	22	16	—	11·1
Miliary tubercle	—	3	2	—	2·7
Caseous ,,	—	2	2	—	

—	1892	1893	1894	Total cases	Per cent. of cases included
III. ABDOMINAL ORGANS (*continued*):					
Kidneys—					
Amyloid	—	11	9	—	6·1
Parenchymatous nephritis	13	3	6	36	6·7
Interstitial "		7	7		
Miliary tubercle	12	11	5	35	6·5
Caseous "		2	5		
Tubercular pyelitis	—	1	—	—	—
Bladder—					
Tubercular ulceration	1	2	1	4	·7
Suprarenals—					
Tubercular	2	1	1	4	·7
Generative organs (male)—					
Tubercular epididymis	3	4	2	9	1·6
" prostate	—	2	2	—	—
" vesiculæ seminales	—	4			
Generative organs (female)—					
Tubercular ovaries	—	2	5	—	—
" tubes	—	2			
IV. CIRCULATORY ORGANS:					
Tubercular pericarditis	—	—	4	—	—
Acute endocarditis	2	—	—	—	—
Chronic "	2	7	11	20	3·7
Congenital malformation	1	2	—	—	—
V. GLANDULAR SYSTEM:					
Bronchial and mediastinal glands—					
Enlarged	—	113	90	—	66
Caseous	—	25	6	—	9·5
Calcareous	—	11	—	—	—
Cervical glands—					
Caseous	—	3	1	—	—
Mesenteric glands—					
Caseous	—	30	6	—	11
VI. MENINGES AND BRAIN:					
Tubercular meningitis	2	8	8	18	3·3
" tumours	—	2	1	—	—
Cerebral abscess	1	—	—	—	—

Bronchi and trachea.—Acute inflammation of the bronchi may be present, and as a rule the lining membrane of the air passages is either intensely reddened or congested, and the tubes contain a quantity of muco-purulent secretion. When the trachea is the seat of tubercular ulceration the change often extends to the main bronchi, appearing either as punctiform erosions or as irregular areas formed by the coalescence of separate foci.

The lining membrane of bronchi which are in direct communication with cavities, or which pass through pneumonic areas, is often intensely injected and swollen, and may have undergone extensive tubercular ulceration.

	No. of cases in which lesion was present
Duodenum	7
Jejunum	68
Ileum	192
Cæcum	193
Vermiform appendix	135
Colon	137
Rectum	44

In 382 cases the small intestine was alone affected in 33 cases; the large intestine alone in 63 cases; both small and large intestine were affected in 200 cases.

The large number of cases in which the vermiform appendix was the site of ulceration is remarkable, but a 'perityphlitic abscess,' the result of perforation of the appendix, was only found in a single case.

In 16 cases in which perforation of a tubercular ulcer occurred, the site of perforation was as follows:

Duodenum	2
Jejunum	0
Ileum	7
Cæcum	5
Vermiform appendix	1
Colon	1
Rectum	0
	16

Peritonitis.—Perforation of the intestine may be followed by acute suppurative peritonitis, or chronic peritonitis may be present and the area of inflammation may correspond to the peritoneal aspect of ulcers which may or may not have perforated; or the lesion may be general throughout the peritoneal cavity.

In one of the cases analysed, purulent peritonitis occurred from tubercular ulceration of the intestine without perforation.

Amyloid Disease.—This form of degeneration is of common occurrence in the chronic variety of pulmonary tuberculosis. It was present in 323 cases as follows:

	Cases	Per cent.
Liver	20	6·1
Spleen	38	11·1
Kidneys	20	6·1
Stomach	6	1·8
Intestines	17	5·3

Liver.—Fatty infiltration of the liver occurs in rather more than one-third of the chronic cases; it has been attributed to (*a*) the reabsorption of the fat into the blood during the process of emaciation, and to diminished secretion of bile arising from imperfect digestion, the fat absorbed being in part primarily expended in the formation of bile (Frerichs); (*b*) to an excessive production of easily oxidisable fat by the liver; (*c*) to an accumulation of fat owing to deficient oxidation throughout the body, in consequence of the disease of the lungs.

The exact cause of the accumulation of fat in the liver whilst it is being removed from other parts of the body has not so far been ascertained.

Tubercle of the liver is more common in children than in adults, and in the miliary than the chronic form of the disease.

It is sometimes extremely difficult to recognise the presence of miliary tuberculosis of the liver with the naked eye, and when, as is not uncommonly the case, miliary tuberculosis and cirrhosis are associated, it may be impossible to do so without a microscopical examination.

Spleen.—The spleen is the seat of tubercle in only a small proportion of cases (2·7 per cent.), miliary and caseous lesions occurring with nearly equal frequency.

Kidney.—Tubercle of the kidney in association with the same disease in the lungs usually appears in the form of small granulations in the cortex and pyramidal portions. More extensive infiltration may be present, and caseation may have occurred, but the large 'scrofulous' kidney is a rare complication of chronic pulmonary tuberculosis, although pulmonary lesions are often present at a late stage of that affection.

Amyloid degeneration is the most common renal lesion. It will be seen from the above table to have been present in 6·1 per cent. of the cases examined. No direct relation exists between the other forms of chronic Bright's disease and the pulmonary affection.

Generative organs.—Tubercular disease of the generative organs in the male is rare as a complication of the pulmonary affection, but the epididymis and vesiculæ seminales should always be carefully examined in cases of miliary tuberculosis in which the source of infection is doubtful.

Chronic caseous disease of the ovaries and Fallopian tubes was met with in 2·7 per cent. of the cases analysed.

Organs of circulation.—The subject of tuberculosis of the pericardium is considered in the chapter on general Serous Membrane Tuberculosis (*vide* p. 587).

Chronic valvular lesions were met with in 3·7 per cent. of the Brompton Hospital cases, the mitral valve being most frequently affected (17 times in 20 cases). In 500 cases analysed by Dr. Percy Kidd, 27 examples of chronic endocarditis were found (5·4 per cent.). Cases of congenital stenosis of the pulmonary orifice not infrequently develops tubercular disease of the lungs.

Glandular system.—Enlargement of the bronchial glands is commonly present (66 per cent.), and in a considerable number of cases these, and also the mesenteric glands, are either caseous or calcareous.

Meninges and brain.—Tubercular lesions of these organs are more common in childhood and as a part of general miliary infection than as a complication of chronic pulmonary tuberculosis. It is indeed somewhat remarkable that tubercular meningitis should rarely occur (3·3 per cent.).

CHAPTER XXXIV

CHRONIC PULMONARY TUBERCULOSIS

(continued)

SYMPTOMATOLOGY—PHYSICAL SIGNS—DIAGNOSIS—PROGNOSIS

	PAGE
Symptomatology	364
Modes of onset	364
Symptoms	366
Examination of expectoration	366
Respiration	369
State of the blood	370
Pulse	371
Digestive system	371
Physical signs	372
Of tubercular infiltration	373
Of softening	375
Of excavation with contraction	376
Diagnosis	377
Pseudo-tuberculosis	379
Prognosis	379

Modes of onset.—This varies much in different cases. The following clinical types may be recognised:

1. *Insidious.*—This is the most common form in which the disease appears. The patient is either unable to fix definitely the time when his health became affected, or perhaps dates the attack from a cold of no unusual severity or from some illness in which the lungs were not apparently involved. Of late it has been extremely common to find the following statement in the clinical history of cases of pulmonary tuberculosis: 'The patient suffered from an attack of influenza in ——, and states that he has never felt well since that time.' There is a history of gradual failure of strength, anæmia, cough, expectoration, and emaciation. Sweating may have been present at night, and on exertion the breath has probably been noticed to be short. Loss of appetite, dyspepsia,

and vomiting after food often form a prominent feature in the history of such cases. The tongue may be furred, with prominent red papillæ. If the temperature has been taken in the afternoon or evening, it will probably have been from one to two degrees above the normal.

2. *Bronchial catarrh followed by emphysema.*—The cases presenting this mode of onset are scarcely less numerous than the former. There is usually a history of repeated attacks of bronchial catarrh or of acute bronchitis; cough may have been present in the winter for some years, and there may have been attacks of dyspnœa, with wheezing. Expectoration has probably been profuse, and perhaps occasionally tinged with blood. The date of the tubercular infection may be ill-defined or marked by emaciation, loss of strength, an alteration in the character of the cough, and often by the occurrence of night sweats. In many cases presenting this mode of onset, the cough, which has previously disappeared in the summer, persists during that period of the year, and the more grave symptoms are first noticed in the following winter.

3. *Pleuritic onset.*—In these cases the appearance of the disease has been preceded by one or more attacks of pleurisy, either dry or with effusion; if there has been effusion the fluid may have been completely absorbed. In some cases there is a history of pleurisy with effusion affecting first one side of the chest and after a varying interval the other, and after both attacks complete absorption of the fluid may have taken place.

The immediate onset of the pulmonary lesion is generally marked by cough, emaciation, pyrexia, and night sweats. In such cases, as in those presenting the modes of onset previously described, there is often a history of tubercular disease in the parents or in other members of the family.

4. *Hæmoptoic onset.*—In a certain proportion of cases the health is apparently but little affected prior to the occurrence of an attack of hæmoptysis. This attack may be immediately followed by the ordinary symptoms and signs of the disease, or, as not uncommonly happens, the attack is quickly recovered from, and on examination of the chest shortly afterwards, few, if any, definite lesions may be detected. There may be a considerable interval before another attack of hæmorrhage occurs, but after a time the ordinary symptoms of the affection appear. It is almost certain that in many cases of this kind there is an old tubercular lesion of the lung, and that the attack of hæmoptysis apparently marking the onset of pulmonary tuberculosis has really been preceded by symptoms which have either escaped the notice or the memory of the patient. Cases presenting this mode of onset are not uncommon in young men whose health has been impaired by overwork, and in men beyond middle age in whose family there is no history of tubercular disease.

5. *Laryngeal onset.*—Although in the vast majority of cases the larynx is involved secondarily to the lungs, the organs are sometimes attacked in the reverse order. On this point few who have had

extensive experience of the disease are in doubt, although the fact is admittedly difficult of proof in any given case, as the absence of pulmonary signs does not imply the absence of lesions, and such signs may be masked by the changes in the larynx. It is, however, quite certain that when tuberculosis is present in both situations, the laryngeal affection may be severe and advanced at an early period, long before it usually makes its appearance.

The first symptoms complained of are irritable sensations in the larynx, mucoid expectoration, and cough; subsequently the voice becomes hoarse and husky, and at a later period may be completely lost. When this condition is suspected to be present, the sputa, or some of the secretion removed from the larynx with a brush, should be examined for bacilli, as they may be present in the expectoration before the appearance of any signs of pulmonary disease.

Symptoms.—The early symptoms are included in the foregoing description of the modes of onset of the disease. Of those which attend its course, *cough* is the most prominent and the one which chiefly attracts the patient's attention. It also constitutes, and perhaps not unnaturally, his guide as to the efficacy of treatment. Cough is usually present at an early period of the illness, and throughout its course is rarely absent for any lengthened period, except in cases of complete arrest. At first short, dry, and 'hacking,' it is soon attended with expectoration, and assumes the character observed in bronchitis. It may be present at intervals throughout the day, but is commonly most troublesome after meals or unusual exertion, and on lying down at night and on rising in the morning.

Paroxysms of cough, attended by difficulty in expectoration, frequently give rise at more advanced stages to vomiting and leave the patient very 'short of breath' and much exhausted. Cough is often followed by vomiting, especially if severe and paroxysmal in character, as is often the case when cavities contain secretion which is expelled with difficulty owing either to rigidity of the cavity wall or to the contraction of the bronchial orifices. Pressure upon the lower part of the trachea or main bronchi by enlarged bronchial glands is a not uncommon cause of paroxysmal cough, especially in children.

The most important points to notice with regard to the cough are, whether it is 'effective' in producing the discharge of secretion, or only 'irritative' in character, and, so to speak, useless; on this difference the treatment of the symptom mainly depends.

Expectoration, as a rule, occurs early in the disease; it is rarely absent when symptoms have been present for as long a period as two months. The exceptional cases include examples of miliary tuberculosis, caseous consolidation without softening, and fibroid tuberculosis. When tuberculosis follows chronic bronchitis, expectoration may be profuse and catarrhal in character. It consists at first of clear viscid glairy mucus, but this may, by admixture with saliva, and in some cases independently of this, acquire a watery character; subsequently small grey or greenish purulent masses appear. Tubercle bacilli are most often found in these purulent masses, but they may be present in mucoid expectoration.

With the advent of softening the sputa become more profuse, mucopurulent, and yellow or greenish in colour. When the expectoration consists mainly of flattened purulent masses of a greenish-yellow colour which do not coalesce in the spitting-cup, it is said, from the similarity in appearance to a coin, to be 'nummulated.' Such sputum as a rule comes from cavities, but the secretion from chronically inflamed bronchi may present this appearance, although it is usually more fluid in character.

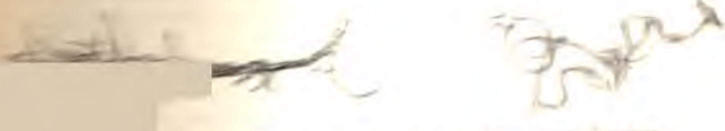
Bayle drew attention to the presence in the expectoration of small opaque white or [illegible]ow masses, which he likened to boiled rice. These are not all [illegible] the same nature (Wilson Fox). Such pellets in mucoid expectoration may be derived from the follicles of the throat, trachea, or bronchi, and possibly also from the alveoli. They consist of mucous and pus cells embedded in a matrix. Small opaque shreddy particles presenting a somewhat similar appearance may be shown, by floating them in water, to consist of elastic tissue fibres and fragments of lung tissue undergoing necrosis. In such masses tubercle bacilli are very likely to be found.

The quantity expectorated daily depends chiefly upon the activity of the disease. When rapid destruction of the lung is in progress six to ten ounces or even more is by no means an uncommon quantity. Patients usually describe the sputa as either salt or sweet in taste; the former is present in the early period, the latter when the expectoration has become purulent.

The sputa, when puriform, have a faintly sweet odour. Bronchiectasis, gangrene, and putrid decomposition of secretion in cavities may render it offensive, but it is somewhat remarkable how rarely this occurs.

The presence of blood, either as streaks, stains, or clots, is known from its colour; when recent it is red, but when it has been retained for some time in the bronchi or in cavities it is of a dark purple or blackish tint. Clots of this nature are almost invariably expectorated for some days after an attack of hæmoptysis. The characters of expectorated blood are more fully described in the chapter on Hæmoptysis.

The microscopical examination of the sputa is chiefly undertaken to determine the presence of elastic tissue or tubercle bacilli.



-ELASTIC TISSUE OF THE LUNG FROM THE SPUTUM (× 200) OF A CASE OF TUBERCULOSIS.

e.—This appears as single or multiple, curled, e fibres forming a network usually with traces of the alveolar arrangement, and in cases of rapid softening, portions of pulmonary tissue may be found in which the alveolar structure can be clearly made out.

If some of the thick purulent material is placed upon a glass plate about four inches square and compressed into a thin layer by a second glass plate about three inches square, the elastic tissue on a black background appears to the naked eye as greyish-yellow spots. The fragment may then be removed, placed upon a microscopic slide, and further examined (Andrew Clark).

Another method in common use is Fenwick's. The sputum is boiled for a few minutes with an equal volume of a solution of caustic soda (20 grs. to the ounce), and the fluid is allowed to stand in a tall conical glass for twenty-four hours. The sediment is then examined microscopically. The observer must be careful to use a silk handkerchief to clean the slides and cover-glasses, and not to mistake threads of cotton or squamous epithelium seen edgewise, or other extraneous materials, for elastic tissue, which is characterised by the well-marked outlines and peculiar curve and the alveolar arrangement of the fibres, and their occurrence, for the most part, in groups. The alveolar arrangement of the fibres is the only trustworthy appearance.

Elastic fibres from bronchial ulcerations are said to be fragmentary and to lack the curled appearance of those from the alveoli.

The presence of elastic tissue in the sputa is absolute proof that a destructive disease of the lung is in progress, and in the great majority of cases in which elastic tissue is found the disease is tubercular.

The test is, however, not perhaps so much used now as formerly, owing partly to the difficulty in finding the fibres when the expectoration is profuse, and to their frequent absence from the opaque masses; but chiefly to the more complete information obtained from the discovery that the sputa contains tubercle bacilli.

Tubercle bacilli are found in the sputa in the great majority of cases of the disease, and they should be sought for in every case in which there is any reason to suspect its presence.

If a systematic examination of the sputa for bacilli be made in every case of chest disease of any importance, the observer will certainly meet with many cases in which they are absent, although everything pointed to the likelihood of their presence, and many also upon which their unexpected discovery throws quite a new light.

Repeated examinations should be made before the search is abandoned, as they may be discovered after many failures. They have been found by many observers in the initial hæmoptysis. In purulent secretion from vomicæ, they are almost invariably present, and may be in large numbers. In cases of fibroid tuberculosis, on the contrary, they are often found with difficulty or not at all.

It has been clearly established that there is no constant relation between the number of bacilli in the sputa and the activity of the disease, but when rapid softening is in progress they are usually present in large numbers, and a tendency to quiescence of the morbid process is usually accompanied by a diminished discharge of bacilli, and with complete arrest they may be absent or there may be no expectoration.

The continued presence of bacilli in the sputa does not appear necessarily to imply that the disease is in active progress. In a case under observation of the writer, bacilli have been present in the expectoration on every occasion on which an examination has been made since 1882. The patient has during that period been actively at work, and has generally enjoyed good health; he is certainly as well, if not better, now than he was fourteen years ago.

Various crystals are found in tubercular sputa, also epithelial and pus cells, blood corpuscles, and many organisms, chiefly the various forms of micrococci; but their presence is as yet of little clinical importance as compared with that of tubercle bacilli and elastic fibres.

A diminution in the amount of the sputum and the absence of the purulent character are, speaking generally, signs of lessened activity of the disease, and when complete arrest has occurred there may be either none, or only so much on rising in the morning as is often expectorated by persons who consider themselves to be in good health.

Pain in the chest is a common symptom. It is more often referred to the infra- or supra-clavicular regions than elsewhere, and in such cases usually indicates the presence of disease in the underlying pleura or lung.

Complaint is often made of pain or tenderness about the apex of one lung or over the pectoral region when the morbid process is in a very early stage, even before any objective signs are present, and such a complaint should never be regarded lightly in cases where there is reason to suspect tuberculosis of the lungs from the general symptoms, although trustworthy physical signs are absent. Pain in the lower axillary or submammary regions is generally due to localised inflammation of the pleura, or to the stretching of adhesions during cough.

The contraction of an apex cavity may be accompanied by pain from the changes in the pleura which almost invariably attend such a condition. Very severe pain in the supraspinous region, or at a lower level in the back, may be due to extension of the disease through both layers of the pleura, previously adherent, and the formation of a burrowing sinus in one or more intercostal spaces. Such a condition is not uncommonly present post mortem in acute cases.

The importance of pain as a symptom of the occurrence of pneumothorax is emphasised in the chapter on that subject (*vide* p. 633).

Respiration.—The breathing is quickened in proportion to the area of lung involved and the rapidity with which the disease is extending, but in almost all cases the rate is above the normal.

As a rule it increases in frequency with the evening rise of temperature, but there is a less definite correspondence between the respiration and the degree of fever than between the former and the pulse rate. The variations between the rate of the pulse and the frequency of respiration are, however, so extreme that the observation of the pulse-respiration ratio is of comparatively little value.

State of the Blood.—The state of the blood in pulmonary tuberculosis has been investigated by Stein and Erbmann.[1] Leucocytosis has been observed in tuberculosis under the following conditions:

1. Cavities in the lung.
2. Inflammatory processes at the end of a case of phthisis.
3. Chronic suppuration following on caries.
4. Tuberculous hyperplasia of glands without much destructive change in the lung.

The authors found that in the early stages and with limited lesions leucocytosis was absent. Leucocytosis increased with the occurrence of softening in tubercular foci. When fibroid lesions predominated leucocytosis was absent. Leucocytosis appears after hæmoptysis (as after all forms of hæmorrhage), but subsequently disappears.

They ascribe the occurrence of leucocytosis, not to the action of the specific organism of tuberculosis, but to a secondary infection by various virulent bacteria and micrococci which leads to the breaking down of the lung. They agree with Maragliano that the phenomena of the 'hectic' stage of tuberculosis have nothing to do with tuberculous infection, but are to be ascribed to the action of streptococci, staphylococci, and other organisms. The writer has expressed his dissent from this view elsewhere (*vide* p. 324).

Cabot's[2] conclusions as to the state of the blood in tuberculosis are as follows:

1. The red corpuscles are usually normal.
2. The hæmoglobin is diminished.
3. Both red corpuscles and hæmoglobin are in some cases diminished.
4. The leucocytes undergo no changes in character.

As to the occurrence of leucocytosis in pulmonary tuberculosis, his statements agree in the main with those of Stein and Erbmann.

1. In the early stages the white corpuscles are normal.
2. After an attack of hæmoptysis they are increased.
3. If cavities are present, there is no leucocytosis; if increased, there is no cavity.
4. With extensive infiltration of the lungs (tubercular pneumonia) there may be marked leucocytosis, but this is not invariable.
5. In fibroid tuberculosis there is no increase of leucocytes.

---

[1] *Deut. Archiv. für Klin. Med.* December 1895.

[2] *Yearbook of Treatment*, 1897, p. 45, Dr. Schorstein.

6. If fever is absent there is no leucocytosis. If pyrexia is due to the presence of pyogenic organisms, there is an increase in the white corpuscles; if not, there is no leucocytosis.

Dyspnœa.—In cases of chronic tuberculosis the patient generally admits that he is 'short of breath' on exertion, but there is rarely a subjective sensation of dyspnœa until towards the end of the disease, unless emphysema has preceded tuberculosis. Urgent dyspnœa, apart from evidence of very extensive disease, should always suggest the presence of miliary tuberculosis of the previously unaffected parts of the lungs.

Patients of the neurotic type often present this symptom in an unusual degree.

Pulse.—An increase in the pulse rate is generally present when the disease is extending and there is considerable pyrexia, but the frequency of the pulse is influenced more by the strength of the patient than by the degree of fever (Wilson Fox). As a rule, however, a slow pulse is associated with a moderate degree of pyrexia.

The pulse rate falls after 9 P.M. and rises generally in the morning after 5 A.M. (Edward Smith). Wilson Fox's observations show that the quickest pulses were usually observed in the morning.

In the later stages of the chronic disease the pulse is usually very rapid and of low tension, and attacks of pseudo-angina and of palpitation are common. The latter are especially observed in patients with marked retraction of the left lung (Powell).

A rapid pulse with a low temperature is usually a sign of exhaustion.

Emaciation.—So long as the morbid process continues active the patient, generally speaking, loses weight, but it is not uncommon in hospital patients for a moderate increase of weight to accompany for a time an extension of the disease. This is a natural result of more abundant food and improved surroundings, and usually only continues for a short period. Should, however, no favourable change then occur, the weight previously gained is lost and emaciation progresses.

Quiescence of the morbid process or its arrest is usually followed by an increase of weight.

Loss of weight may take place, although pyrexia is absent, in chronic cases attended by a failure of vital power.

Digestive system.—The onset of the disease is frequently accompanied by loss of appetite, thirst, and signs of gastric catarrh, and during its course disorders of digestion generally attend periods of increased activity of the morbid process. As a general rule, if the appetite is good, digestion is unimpaired, and *vice versa*.

The tongue is often covered with a thin fur and dotted with red papillæ, or a thin white fur may be present over certain areas, whilst the remainder of the tongue is clean and the mucus membrane looks red and irritable. When gastric symptoms are prominent the tongue may be thickly coated. An atonic condition of the digestive organs is usually accompanied by a large pale flabby tongue with its edges marked by the teeth.

In the later stages of the disease, when there is continuous pyrexia of a remittent type, and when possibly ulceration of the intestine is present, the tongue is often red, raw, dry, and fissured. When the tongue is completely free from fur, red, and deeply marked by transverse fissures, it is important to ascertain that this is not, as it may be, its normal condition, as, owing to such a possibility having been overlooked, we have known very erroneous inferences drawn.

Vomiting is a common symptom in the later stages of the disease, but it may occur at quite an early period, even before the appearance of any well-marked physical signs in the lungs. When, as is frequently the case, it only occurs after a severe paroxysm of cough, it may be unconnected with any disorder of digestion; but during periods of active change in the lungs vomiting is apt to occur shortly after meals, and may then possibly be due to an irritant lesion of the pneumogastric nerve.

The early stages of the disease and periods marked by digestive disturbances may be attended by constipation, but this is not as a rule a marked symptom.

Diarrhœa may appear early in the disease, and in some cases it occurs during its course from slight causes, such as errors in diet, and passes off under appropriate treatment. When, however, as is more commonly the case, it first appears at a late stage of the disease, it is generally an indication of the presence of ulceration of the intestine. It is not necessarily proportionate in severity to the extent of the lesions, and may be absent in cases attended by widespread ulceration of both the small and large intestine.

When there are signs of the presence of amyloid disease of other viscera, diarrhœa may be due to a similar condition of the intestine.

Pain and tenderness on pressure may accompany ulceration of the bowel, but it is rarely severe, and may be slight or absent in cases attended by severe diarrhœa.

Hæmorrhage from the bowel is an event of rare occurrence.

Fistula in ano occurs far more frequently in males than females, being indeed exceedingly rare in the latter subjects. It may precede the development of obvious tuberculosis, but as a rule it forms after the disease has made considerable advance in the lungs.

In such cases it is extremely probable that a tubercular ulcer has preceded the fistula. On reference to the table given on p. 362 it will be observed that tubercular ulceration of the rectum was found in 44 out of 323 cases examined post mortem, or in 13·6 per cent.

PHYSICAL SIGNS

The signs present in the early period of chronic pulmonary tuberculosis must necessarily be interpreted according to our knowledge of its morbid anatomy, although little is certainly known of the exact relation between the pathological conditions and the physical signs which mark this stage of the disease.

Notwithstanding this fact, the anatomical basis is the most convenient for purposes of description, and we shall consider the signs which attend (*a*) the stage of infiltration, (*b*) the softening of the tubercular lesion, (*c*) the formation of a cavity, and (*d*) the contraction of a cavity, in the above order.

Signs attending tubercular infiltration.—**Inspection.**—The chief parts to which attention should be directed are the supra- and infra-clavicular regions. At a very early period nothing definite may be observed, but if the area of the lung affected is of some extent the expansion of the upper part of the chest on the corresponding side is diminished. This is often more noticeable if the observer stands behind the patient and looks over his shoulders, or if, when standing in front of the patient, he places his thumbs upon the second ribs below the clavicles, and fixes his eyes upon his thumbs while the patient breathes deeply.

Palpation.—Changes in the vocal fremitus are of great importance in determining the presence of disease of the apex in an early stage. Beneath the right clavicle the vocal fremitus is normally more distinct than on the left, and the trained hand acquires a standard by which variations from the normal are judged.

If the vocal fremitus is equal on the two sides, and well marked, it is probably increased at the left apex, and a presumption arises that the left upper lobe is the seat of disease. If the fremitus is more marked at the left apex than the right, the presumption that the left upper lobe is affected is still stronger. If, on the other hand, the vocal fremitus beneath the clavicles is equal, but less marked than normal, probably the voice conduction is diminished at the right apex, and the condition of the right upper lobe must be carefully investigated in the further course of the examination.

Percussion.—In the early stage a slight difference in the percussion note may sometimes be elicited by tapping the clavicles, when elsewhere a difference is unappreciable. Resonance may not extend so high above the clavicle on one side as on the other, or a slight difference in the percussion note on the two sides may only be present when the breath is held after a deep inspiration, owing to the lesser amount of air which enters the affected lung. Firm percussion in the supra-spinous fossæ will often elicit a dull note when the evidence obtained from the examination of the front of the chest is doubtful.

Auscultation.—Normally, the respiratory murmur is slightly higher pitched on the right side, and expiration is rather more prolonged; but the breath sounds may be bronchial beneath the right clavicle, when there is no disease at the apex of the right lung. This is a fruitful source of error in diagnosis, one of the most common mistakes of an inexperienced auscultator being the discovery of a cavity at this site when no lesion of any kind is really present.

It is difficult to make an absolute statement as to the condition of the breath sounds at this stage. They are variously stated to be harsher than normal and weaker than normal. There is probably considerable variation in this respect; but, as a rule, the harsh wavy breath sound with prolonged expiration precedes the stage of feeble breathing. In the latter condition there is little difficulty in arriving at a correct diagnosis, as, when the breath sound is feeble, adventitious sounds can usually be elicited by making the patient cough, whereas with harsh, wavy, interrupted breathing râles are generally absent. It may often be noticed that when the breathing above the clavicle and immediately below it is feeble, the breath sounds in the second and possibly in the third interspace present the harsh, wavy, interrupted character. In such cases it is certain that the area of most advanced disease underlies the site of feeble breathing.

In neurotic subjects wavy, interrupted breath sounds do not necessarily indicate disease, and the condition is then bilateral. Conduction of the voice and also of the heart sounds to the apex is facilitated by consolidation of the lung, and this increase of vocal resonance may be present when the area of disease is limited. The presence of a systolic murmur in the subclavian artery is not a sign of much value in doubtful cases.

As the area affected becomes more densely infiltrated, the breath sounds become high pitched and acquire a bronchial or blowing character, which is present both on inspiration and expiration; bronchophony may now be audible. The breath sounds over the posterior apex of the lower lobe may be harsh or bronchial, indicating that infiltration is in progress there.

When the breathing is feeble at one apex it is often puerile over the opposite upper lobe from increased functional activity of the part—a favourable condition, the presence of which should always be looked for. In such cases error as to the site of the disease is not uncommon, the feeble breath sound being overlooked and the significance of the harsh sound misinterpreted.

Increase of functional activity is indicated by puerile breathing, increased expansion, and possibly also by enlargement of the part, whilst a harsh, wavy, interrupted breath sound, from which the vesicular quality is absent, and diminished expansion, are signs of the presence of disease.

Adventitious sounds are, as a rule, absent at the earliest period, the first to appear being fine 'dry' râles, often heard only with deep inspiration, and then limited to a single râle, or perhaps three or four such sounds may be heard, possibly only whilst the patient coughs.

This râle is not so fine as 'crepitation' (the 'fine crepitation' of pneumonia), and when several such râles are present the individual crackles are not separated by equal intervals as in the crepitation of pneumonia. These râles may disappear if the disease becomes arrested at this stage, but as a rule they pass at a later period into sounds of a 'clicking' character, audible both on inspiration and expiration, but more distinct during the former act.

In doubtful cases one should never omit to note the effect of cough in the production of râles. A careful examination of the supra-spinous fossæ is as necessary in all cases as of the supra- and infra-clavicular regions; the signs in this situation often show that the disease is more advanced on the posterior aspect of the lung, and a positive diagnosis may often be made after an examination of this region when the investigation of the front of the chest has yielded doubtful results.

The signs above described may never develop into those which mark the second stage of the pathological process. Post-mortem and clinical experience afford abundant proof of the frequency of arrest of the disease in the stage of infiltration.

When arrest has occurred the breath sounds at the apex are usually weak, and râles are absent. The lesion contracts from the formation of fibroid tissue, emphysematous bullæ are formed, the surface of the lung becomes puckered, and the percussion note more resonant.

There is usually little if any retraction of the upper part of the chest with arrest in this stage.

Softening of the tubercular lesion.—The altered character of the adventitious sounds affords the first evidence that the process of softening has commenced. The râles become more numerous, larger, and more 'liquid.' They are variously termed 'clicks,' medium sized and large crepitant râles, moist crepitation and humid crackle.

Signs of softening may also possibly be detected at the apex of the lower lobe, and the area of infiltration along the interlobar septum will have extended.

The breath sounds are now high pitched, blowing or bronchial, and the vesicular quality may be quite lost.

The vocal resonance is increased, and bronchophony or even pectoriloquy may be present.

The formation of a cavity.—The process of softening gradually merges in chronic cases into that of excavation. A case which has but recently advanced to this stage, and in which fibrous changes are only present to a moderate degree, may present some or all of the following signs:

Inspection.—The point of the shoulder is depressed and the scapula and clavicle are prominent, the supra-clavicular and supra-spinous fossæ are sunken, the pectoral region is flattened, and the first and second intercostal spaces are wide and deep; there is a well-marked difference of expansion on the two sides. With disease at the left apex, the cardiac impulse may be slightly displaced upwards.

Palpation.—Vocal fremitus is markedly increased both in front and in the supra-spinous fossæ.

Percussion.—The note is either high pitched and wooden or has a tubular or tympanitic quality. A tubular note generally indicates that the cavity is surrounded by consolidated lung. A cavity of large size, with thin walls, near to the surface of the lung, may give the cracked-pot sound on forcible percussion during inspiration, the patient's mouth being open at the time.

The percussion note may indicate some degree of enlargement of the upper lobe of the opposite lung.

Auscultation.—The typical breath sound is termed cavernous, the expiratory sound being lower pitched, more hollow, and more prolonged than the inspiratory. It may be metallic, and with a large cavity may present the amphoric character.

In some cases over a large cavity the breath sound is absent, or it may be absent in the first interspace but distinctly cavernous below. During or after cough, however, the nature of the condition usually becomes clear.

The typical adventitious sounds are metallic or coarse gurgling râles; they require for their production air and fluid in the cavity and a patent bronchus, conditions which are usually present in cases in the stage here described. Râles produced in the neighbourhood of a cavity may, however, acquire a metallic or 'echoing' character. With a thin-walled cavity the post-tussive suction sound may be heard.

Evidence of excavation may also be found at the apex of the lower lobe of the lung primarily affected, and of still more extensive infiltration with softening in that lobe.

Pectoriloquy of both varieties is common, but it may be here remarked that whispering pectoriloquy is one of the most fallacious signs of the presence of a cavity.

Signs of excavation with contraction.—The signs of a chronic contracted cavity at the apex of one lung, with induration around and marked thickening of the pleura, are as follows:

Inspection.—The upper part of the chest is usually much contracted, and there may be some degree of curvature of the spine. Expansion may be absent; the interspaces are depressed. In disease of the left upper lobe the heart may be markedly displaced upwards and slightly outwards, and the impulse may be seen and felt in the third left interspace or even higher. If the lower lobe is also contracted, the apex beat may be in the left axilla. With contraction of a cavity in the right upper lobe the heart may be displaced to the right, and may lie wholly to that side of the sternum. The displacement is less marked if, as sometimes occurs, there is great compensatory enlargement of the right middle lobe. With a contracted cavity at the left apex, pulsation may be observed over the cardiac area, owing to the heart having been either uncovered or displaced, and also close to the sternum in the second and third left interspaces from uncovering or displacement of the pulmonary artery, or uncovering of the auricular appendix.

Percussion.—There is high-pitched wooden dulness over the site of disease from thickening of the pleura and induration of the lung, and marked increase in the sense of resistance. The enlargement of the opposite upper lobe may be shown by the fact that there is resonance on percussion for two inches or more beyond the mid-sternal line towards the diseased side.

Auscultation.—There may be complete absence of breath sound in front, but if the arm is raised and the end of the stethoscope placed at the apex of the axilla cavernous breathing will generally be detected. The cavity will probably be dry, and there may be no adventitious sounds. If there are any they are of a metallic quality.

The impulse of the displaced heart may cause a systolic 'air-wave' murmur in the cavity (*vide* p. 67), and the murmur may be audible in the trachea or mouth, or even at a distance from the patient when his mouth is open.

With a contracted cavity at the left apex, a loud systolic murmur, due either to displacement and compression of the pulmonary artery or to narrowing of the subclavian artery, may be heard in the first and second left interspaces. The writer has known such a case to be regarded for some time as one of valvular disease of the heart.

There may now be evidence of contracted cavities surrounded by indurated tissue in the corresponding lower lobe.

If the change in the opposite lung is chiefly emphysematous, the breath sound over the upper lobe will be weak; if, on the other hand, as is commonly the case, it is in part at least hypertrophic and associated with increased functional activity, the breath sounds there will be harsh and expiration will be prolonged.

DIAGNOSIS

The recognition of the disease has been much facilitated by the discovery of the bacillus tuberculosis. In all cases of doubt the diagnosis is greatly influenced by the results of the examination of the sputa.

The presence of tubercle bacilli in the sputa is conclusive, and now that we are provided with an absolute positive test of the tubercular nature of a pulmonary lesion, the use of this method of examination should be considered necessary in all cases, no matter how clear may be the indications derived from the clinical history and the physical examination of the chest.

In some cases repeated examinations of the sputa are necessary, as bacilli may be absent at some period in all forms of the disease, except perhaps in cases accompanied by active softening.

In a case of extensive disease of the larynx, considered for some time by most competent observers to be certainly tubercular—a view the correctness of which was rendered extremely probable by the presence of obvious signs of a lesion at the apex of the right lung—repeated examination of the sputa for tubercle bacilli gave negative results. The use of this test had not at first been considered necessary, as the import of the physical signs was thought to be unmistakable. Subsequently the syphilitic nature of the disease was rendered at any rate extremely probable by a very marked improvement immediately following the administration of iodide of potassium. The patient ultimately recovered completely, physical signs of some induration alone remaining at the right apex; but a moderate degree of laryngeal stenosis was produced, and will probably be permanent.

In a case of fibroid tuberculosis of the left apex under the care of the writer no bacilli were found in the sputa on repeated examinations extending over a month during the first stay of the patient in hospital; a few were then discovered. On subsequent admission after an interval of nearly a year tubercle bacilli were found at the first examination, whilst during a third stay in hospital of nearly three months' duration they were absent.

If, on repeated examination of the expectoration, no tubercle bacilli are found, an exact diagnosis may not be possible, but, as just stated, the condition is not inconsistent with the presence of a fibroid lesion. If, however, there is evidence of extensive destructive disease of the lungs, accompanied by the formation of large cavities, and the sputa are free from bacilli, it is almost certain that the disease is not tubercular. The Museum of the Brompton Hospital contains many specimens illustrating the occurrence of such lesions as a result of non-tubercular disease. This subject is discussed in the chapter on Pulmonary Syphilis (*vide* p. 429).

After a primary attack of hæmoptysis there may be no signs sufficiently definite to indicate the site of the lesion, and yet the disease may be proved to be tubercular by the discovery of bacilli in the expectorated blood. More often, however, the expectorated blood in such cases does not contain bacilli; under these circumstances, or when the blood has not been examined for tubercle bacilli, patients are often told that the blood has come from the back of the throat or elsewhere, and cannot have come from the lungs, as there are no signs of disease to be discovered in those organs—a diagnosis the correctness of which is rarely borne out by the subsequent history of the case.

The diagnosis may be attended with difficulty when the symptoms suggest tubercular disease, but the physical signs are of doubtful import and there is no expectoration.

The cases in which the above conditions are fulfilled are very often delicate-looking young women suffering from anæmia. In such cases, and in all others included in this category, it is a golden rule never to make a diagnosis of tuberculosis from doubtful physical signs, and particularly not from a single sign. The examination of the chest must be systematic; the evidence obtained on inspection and palpation must be considered with and must agree with that derived from percussion and auscultation. When the signs, separately considered, are of doubtful import, but are yet in harmony and all point in one direction, a positive opinion may be given. But the observer must beware of relying upon auscultation with its many fallacies, and a definite opinion should rarely be given in doubtful cases unless a record of the morning and evening temperature for at [illegible] a week is available. The absence of pyrexia is not conclusive, but it tells against a diagnosis of tubercular disease.

When there is clear evidence of a pulmonary lesion, but expectoration is absent, or no tubercle bacilli can be found in it on repeated examination, the exact site of the lesions is, as we have already pointed out, a factor of great importance in estimating the probability of the disease being tubercular.

As, however, primary basic tubercular disease, although extremely rare, does occur, any particular case may happen to be of this nature.

Pseudo-tuberculosis.—The use of this term, which appears to be gaining ground, is, we think, to be deprecated. It is applied to diseases which either present a superficial likeness to pulmonary tuberculosis or produce lesions similar to those of tubercle. A natural result of the systematic examination of the expectoration for tubercle bacilli, which we have long foreseen and which has mainly influenced the writer in insisting on the necessity of so doing, and on the use of the term 'pulmonary tuberculosis' as opposed to 'phthisis,' is the discovery that other agents besides the tubercle bacillus are capable of producing destructive disease of the lung, and also of lesions elsewhere which somewhat resemble those of tubercle. This is, however, in our opinion, no justification for the use of such a term as 'pseudo-tuberculosis.' It is an incentive to discover the nature of such diseases and to adopt a terminology based on etiological grounds. This point is further considered in the section dealing with diseases due to the presence of moulds and fungi (*vide* p. 459).

PROGNOSIS

The prognosis in cases of the miliary, caseous, and fibroid varieties of the disease has already been described.

In chronic pulmonary tuberculosis a prognosis is formed by a careful consideration of the following points :

1. The stage of the morbid process.
2. The rate of progress in relation to the duration of the symptoms.
3. The condition of the unaffected lung.
4. The character of the general symptoms.
5. The presence of complications.
6. The constitutional state and family history, including the age and sex of the patient.

1. **The stage of the morbid process.**—That arrest most commonly occurs in the stage of infiltration is abundantly proved both by clinical experience and by the nature of the obsolete lesions found on examination post-mortem of persons dying from non-tuberculous disease.

In nearly 2,400 autopsies at the Middlesex Hospital obsolete tubercular lesions were found in the lungs in 219 or 9 per cent.[1]

---

[1] 'Arrested Pulmonary Tuberculosis,' J. K. Fowler.

In 176 cases the lesion was either fibroid and pigmented, or caseous or calcareo-caseous. In 43 cases the word 'cavity' occurs in the report. It does not, however, follow that in all these cases it was, or had ever been, a cavity recognisable by methods of clinical examination. In many cases so described the appearance of excavation is produced by the growth around a caseous mass of a fibrous capsule, which becomes smooth within—a condition which is often described in post-mortem reports as a cavity with blackish-grey caseous contents. The complete cicatrisation and obliteration of a cavity, recognised as such during life, must be an event of very rare occurrence. The writer has never met with an example of such a condition.

Arrest in the cavity stage may be complete and life may be indefinitely prolonged. The writer is acquainted with a lady who has at the left apex a well-marked cavity which was almost certainly formed more than fifty years ago. As a rule, when arrest has occurred after the formation of a cavity, death, although possibly occurring after a very long interval, is due to tuberculous disease or to some lesion incidental to its presence.

In cases marked in the early stage by an acute course the evacuation of the necrosed tissue is often followed either by decided improvement or a complete arrest of the disease.

Arrest of the disease may occur after the lower lobe has become affected, and the writer has made an autopsy on a case in which complete arrest had taken place after a cavity had formed in that situation.

As a rule, however, when a cavity has formed at one apex and there is extensive infiltration of the lower lobe on the same side, say for a distance of five inches along the line of the interlobar septum, the disease does not undergo complete and permanent arrest, although for long periods there may be little or no evidence of its extension. Such cases not uncommonly develop the characters of the very chronic type already described, the cavity becoming dry and surrounded by indurated lung and the pleura undergoing extreme thickening.

The presence of a lesion at both apices is quite consistent with the occurrence of arrest; it is, in fact, more common post-mortem to find lesions at both apices than at one only. The writer's results in 177 cases of obsolete tubercle were as follows:

Right lung	35
Left "	36
Both lungs	106
	177

Dr. Sidney Martin[1] in 42 cases obtained similar results:

Right lung	12
Left "	9
Both lungs	21
	42

---

[1] *Brit. Med. Journal*, Oct. 31, 1891.

As it is contrary to clinical experience to find both apices simultaneously attacked and for the lesions to be equally distributed through the upper lobes, it follows that in such cases one lung is affected after the other and probably after arrest has taken place.

The special danger which attends the condition of quiescence or arrest in the cavity stage is the formation and rupture of a pulmonary aneurism.

2. **The rate of progress in relation to the duration of the symptoms** is a very important factor in prognosis in the early stage of pulmonary tuberculosis.

Signs that softening is in progress in the upper lobe and that the lower lobe is extensively infiltrated, the disease having been present for only a brief period, are necessarily of unfavourable import. But, again, it must be stated that many cases of an acute or subacute character at the outset subsequently undergo a favourable change, and a period of quiescence if not of arrest follows.

3. **The condition of the unaffected lung** should be carefully noted in all cases. Enlargement accompanied by signs of increased functional activity of the upper lobe of the opposite lung are of decidedly favourable import, and their absence has a contrary significance. Early involvement of both lungs usually implies a rapid course; but exceptions to this rule, as indeed to nearly all others with regard to prognosis in this disease, are not uncommonly observed.

4. **The character of the general symptoms.**—The state of the digestion and appetite, upon the maintenance of which nutrition necessarily depends, are factors of great importance in prognosis. When from almost every other aspect a case is unfavourable we may still be hopeful of a patient who has a good appetite and an unimpaired digestion, as in such cases a period of quiescence usually arrives sooner or later, and even should the course be progressive it will almost certainly be slow.

It is rarely wise to express a positive opinion as to the immediate outlook without a knowledge of the course of the temperature, and the longer the period over which the observations have extended the better. The significance of the various types of fever as regards the character of the morbid process has been considered in the chapter on Pyrexia in Tuberculosis (p. 323).

The most unfavourable condition in a chronic case is the sudden occurrence of continued pyrexia with very slight remissions, as it usually indicates the presence of acute miliary tuberculosis of parts of the lungs hitherto unaffected.

The occurrence of pyrexia of the inverse type at an advanced stage of the disease—a point to which far too little attention has hitherto been given—is of hardly less unfavourable import. Of thirty cases of tuberculosis of the lungs, mostly of the chronic variety, under the care of the writer in the Brompton Hospital, in which this type of pyrexia was observed, fourteen died in hospital [illegible] one immediately after returning home; eight left the hospital [illegible], some of them decidedly so (of these, all had lost weight);

five were either 'relieved' or about the same, but of these three had lost weight, and in the other two the gain of weight was trifling (½ lb. and ¾ lb.). Of the thirty cases only two improved, and in one of these the morning temperature was higher than the evening on two occasions only; in the other the condition was of temporary duration, and was observed on eight occasions.

A high evening temperature with morning remissions stands next in point of gravity. Moderate pyrexia with a normal or only slightly subnormal morning temperature is of favourable import.

A complete absence of pyrexia associated with a gain of weight, diminished cough and expectoration, fewer bacilli in the sputa, and physical signs indicating quiescence of the disease together constitute the most hopeful condition.

If the general state of the patient, the course of the temperature, and the gain or loss of weight are considered as a whole, it is not difficult to form a trustworthy prognosis for the immediate future. Error generally arises from undue attention having been given to a single aspect of the case.

The significance, for example, of a low temperature depends entirely upon the general state. If this is one of exhaustion, the condition is very unfavourable. The physical signs, again, may on the first examination appear to indicate the presence of rapid softening with extension of the disease; but this may be shown by subsequent observation of the temperature, and by a progressive gain of weight, to have been a mistaken inference.

Perhaps no single element in prognosis is of greater importance than the condition of the appetite and digestion.

5. **The presence of complications.**—*Laryngeal tuberculosis.*—In cases presenting this complication the prognosis is generally very unfavourable. As a rule it appears late in the course of the disease.

The use of lactic acid has certainly rendered the outlook in such cases somewhat less unfavourable, and, if employed at an early stage, cicatrisation of an ulcer of limited extent may be hoped for from this mode of treatment. Spontaneous cicatrisation of a tubercular ulcer of the larynx may, however, occur, but such cases are very rarely met with.

In the very rare cases in which the affection of the larynx is either primary or out of proportion to the extent of the pulmonary disease the prognosis is even more unfavourable. It is said, however, that some cases in which the laryngeal affection, although not primary, appears early in the disease run a protracted course.

Hæmoptysis.—The immediate gravity of this symptom depends very much upon the stage of the morbid process at the time of its occurrence. In that of excavation a profuse hæmoptysis is of very serious import, as it suggests the presence of a pulmonary aneurism. In 283 cases of profuse hæmoptysis analysed by Dr. Theodore Williams, death was directly due to the hæmorrhage in 63, the percentage of deaths in the progressive stages of infiltration, softening, and excavation being 13·95, 24·61, and 67·74 respectively.

The influence of hæmoptysis upon the duration of life in chronic tuberculosis depends in a similar manner upon the stage of the morbid process at the time of its occurrence. In the first stage the effect is but slight, whilst in the second and third it is very appreciable.

Pneumonia, using the term in the sense in which we have employed it elsewhere in this work—viz. to indicate the disease usually termed acute pneumonia or lobar pneumonia—is in our experience a very rare complication of the chronic form of the disease, but the occurrence is attended with considerable danger to life. Recovery with resolution may occur; but, if softening of the consolidated area follows, the downward progress is greatly accelerated.

Bronchitis, especially if the smaller tubes are affected, is very liable to be followed by broncho-pneumonia, and in cases associated with extensive emphysema, or in which but little healthy tissue remains, it may prove fatal.

Pleural effusion.—It is difficult to state in anything like precise terms the effect of pleural effusion upon the course of chronic pulmonary tuberculosis. Osler[1] is of opinion that the early implication of the pleura gives 'a stamp of chronicity to the case.' Possibly this may be due in part to the adhesion of the surfaces preventing the occurrence of pneumothorax.

A sero-fibrinous effusion, even if tubercular, as it commonly is, may be completely absorbed, but the occurrence of a purulent or an hæmorrhagic effusion is certainly of very unfavourable import. It must be remembered, also, that in cases of sero-fibrinous effusion of tubercular origin, the infection of the pleura may be only a part of general miliary tuberculosis.

The relation of the various forms of pleural effusion to pulmonary tuberculosis is considered in the chapter on Pleurisy.

Diarrhœa.—Severe diarrhœa often precedes the fatal termination, and whether due to ulceration of the intestine—the most common cause—or to lardaceous disease, the condition is almost equally unfavourable from the point of view of prognosis.

Albuminuria.—The importance of the presence of albumen in the urine as a guide to prognosis depends entirely upon the cause of the condition. On reference to the table in which the lesions associated with pulmonary tuberculosis are given (*vide* p. 358), it will be seen that amyloid disease of the kidneys was found in 6·1 per cent., parenchymatous or interstitial nephritis in 6·7 per cent., and either miliary or caseous tubercles in 6·5 per cent. of the cases. If the albuminuria is due to the occurrence of any one of these lesions in an advanced stage, it is a sign of grave import. In order to determine its cause, the urine must be further examined microscopically for casts and for tubercle bacilli.

It may be mentioned here that the discovery of tubercle bacilli in the urine is much facilitated by the use of the centrifugator. The quantity of urine passed, the amount of albumen present, the specific gravity of the urine, the nature of the casts (if any are found), the occurrence of degenerative changes in the vessels, and of hypertrophy of the heart, and of evidence of amyloid disease of other organs, are points which need careful consideration before an opinion can be given as to the cause of the albuminuria. That it is not necessarily of grave import is shown by the fact that in a case reported by Dr. Williams the patient was living eight years after albumen was found in the urine.

---

[1] 'Tuberculous Pleurisy,' *The Shattuck Lecture*, p. 41.

Pneumothorax.—This is of far more frequent occurrence as a complication of caseous tuberculosis than of the chronic form. The condition of the opposite lung is the chief guide in prognosis. If this is healthy or only slightly diseased, and the general condition of the patient is good, the air may be ultimately absorbed from the pleura, and the lung of the affected side may re-expand to some extent.

When, however, there are extensive lesions in both lungs, a rapidly fatal termination is the rule. In thirty-nine cases collected by Sir R. Douglas-Powell[1] in which death occurred after pneumothorax, the average duration of life was only twenty-seven days. One patient lived for twelve months, one for four and a half months, and one for four months. Death may occur in as short a period as ten minutes.

Dr. Williams[2] has recorded cases observed in private practice in which the results were much more favourable. In one case life was prolonged for twenty-one years, and in others recovery followed. Even should pyopneumothorax follow, the condition may become chronic. An out-patient in this condition attended under the care of the writer at the Brompton Hospital for several years. His chief object in so doing was to demonstrate the 'succussion splash' and receive the gratuities of the students.

In rare cases the occurrence of pneumothorax leads to arrest of the disease, the condition of collapse of the lung which follows acting as a barrier to the further extension of the lesions, either through the air passages or lymphatics.

The subject is more fully considered in the chapter (*vide* p. 632) on Pneumothorax.

Peritonitis.—The presence of any form of peritonitis as a complication of chronic tuberculosis of the lungs is a very unfavourable condition. The peritonitis may be acute or chronic, simple or tubercular, and either general or localised. Acute general peritonitis due to perforation of a tubercular ulcer of the intestine, or to tubercular ulceration without perforation is the most grave form of this complication. General tubercular peritonitis is frequently recovered from when it occurs as a primary disease, but very rarely when it appears in a case in which the lungs are already extensively affected.

A chronic tubercular peritonitis limited to the pelvis or elsewhere is a less serious complication.

[1] *Op. cit.* p. 164.

[2] *Op. cit.* p. 213.

CHAPTER XXXV

CHRONIC PULMONARY TUBERCULOSIS

(continued)

TREATMENT

THE subject of treatment is considered under the following headings:

1. Prophylactic (p. 385).
2. Prevention of infection (p. 386).
3. General: Food, alcohol, open-air, exercise (p. 389).
4. Specific and antiseptic remedies (p. 396).
5. Tonic remedies (p. 402).
6. Symptomatic, including the treatment of complications (p. 404).
7. Climatic (p. 408).

Prophylactic.—A child of tubercular parentage requires unusual care. It should, if possible, be suckled by a healthy wet nurse, never by the mother, and during infancy and throughout the whole period of childhood special attention must be given to the diet, which should be both abundant and easily assimilated, and consist during the earlier years chiefly of milk, cream, eggs, and meat juice. The child should be weighed at regular intervals, and a record kept of the results; a progressive increase in weight being the best evidence that the food is suitable and that nutrition is well maintained.

Under present conditions it is practically impossible for most people to be sure of the freedom from tubercular infection of the source of their milk supply, and, as already stated, milk is a most active vehicle for the virus. Adults must in such a matter use their discretion; but for children, particularly those of tubercular parentage, all the milk used should previously have been boiled.

Such children should, if possible, be brought up in the country and in a dry bracing air. Their rooms should be well ventilated both by day and night, and they should be much out of doors, and exercises tending to promote the expansion of the chest may be commenced at an earlier age than usual. The clothing should be light but warm, and chiefly of woollen materials. The bath, during infancy and up to the age of about five years, will of course be warm, but after that age the tonic effect of a brief application of cold water may generally be included in the measures which are taken to promote bodily vigour.

Sea air and salt water baths are of special value in these cases, especially when any glandular enlargement is present. Acute diseases, particularly measles and whooping-cough, which are apt to be complicated by bronchitis and broncho-pneumonia, should never in these subjects be regarded as 'childish ailments' requiring little care.

The condition of the throat and naso-pharynx requires careful attention. Enlargement of the tonsils may be followed by changes in neighbouring lymph glands, and through this path the specific virus of tubercle not unfrequently enters the body, whilst adenoid growths impede nasal respiration and diminish the expansion of the thorax.

At a later period of life, a residence at a high altitude, by promoting deep breathing combined with the invigorating effect of pure mountain air, is of great service in developing the resisting power to tubercular infection.

By the adoption of such measures the inherited defect of constitution may be overcome, and the child may grow up to manhood and escape the disease.

These children are often highly intelligent, and therefore suffer no ultimate loss from their education in youth having been considered as of secondary importance to the attainment of a high degree of bodily vigour.

The view that tubercular parents may convey to their offspring a constitution which renders them more prone than others to develop the disease is generally accepted by the laity, but the logical inference that individuals thus affected should not marry is very often set aside on the ground of exceptional circumstances.

Although this liability specially affects children of tubercular parentage, it is certainly not limited to them. The offspring of the feeble-bodied are likely to possess less resisting power than the children of the strong and healthy, and are therefore more prone to become the subjects of tuberculosis. It is the duty of the medical attendant, if consulted, to point out the risk attendant upon the marriage and particularly the inter-marriage of such persons.

Prevention of infection.—The important question of the mode of origin of pulmonary tuberculosis is discussed elsewhere (*vide* p. 304 seq.). As already stated, if the view of its specific nature is accepted, there is no escape from the inference that in the great majority of cases the virus enters the body from without—the exceptions being those cases in which tuberculosis is directly transmitted from parent to offspring. It follows that it is the duty of everyone suffering from the disease to guard against becoming an agent in spreading infection.

This he can do by refraining from expectorating in any public or private place, except into a disinfecting solution or a pocket handkerchief, and by not sending linen soiled by sputa to a laundry without previous disinfection.

In hospitals for consumption there is little difficulty in framing and carrying out regulations by which the occurrence of infection can be effectually prevented, and it should not be thought that too much is asked of tubercular patients if both in public and private they are expected to adopt similar precautions.

The following are the regulations adopted at the Brompton Hospital with a view to prevent the spread of infection :

Sputum.—(*a*) Each patient is provided with a spitting-cup, and similar cups are placed on the staircases, and in the passages, out-patient department, and garden. Each spitting-cup contains one ounce of carbolic acid solution (1 in 20).

(*b*) All knives, forks, spoons, and cups used in the wards are washed thoroughly with hot water to which washing soda has been added, and are then subjected to the action of boiling water for at least two minutes.

(*c*) Handkerchiefs of a distinctive pattern are supplied to the in-patients, and are destroyed after use. No patient is allowed to use any other than a hospital pocket handkerchief. Each patient is provided with at least one clean pocket handkerchief daily.

(*d*) All linen soiled with tuberculous matter is kept separate, and boiled in a vessel reserved for that purpose.

(*e*) The sputum is disposed of as follows: the spitting-cups are collected twice daily and removed in covered metal receptacles, specially provided for that purpose, to the place where the sputum is destroyed.

For the destruction of the sputum by heat, and also of the general refuse of the hospital, which may contain tuberculous matter, an automatic 'Destructor' is used. This consists of a chamber the interior of which can be raised to a white heat.

Each spitting-cup, after having been emptied into a vessel, is immediately placed in a vessel containing carbolic acid solution (1 in 20).

After all the spitting-cups have been emptied, they are placed for at least two minutes in water which is kept boiling, or are subjected to the action of super-heated steam. They are then cleaned and returned to the galleries.

Printed directions describing the measures to be adopted to prevent infection are placed in the wards and out-patient rooms, and handbills giving information of a like character are given to out-patients at the discretion of the Assistant Physicians.

Rules to be observed by Nurses

The following are the rules to be observed by nurses in the wards:

1. Nurses are immediately to report to the Sister-in-charge any patient whom they observe spitting upon the floor, or in the fireplaces or washing basins, or elsewhere than in the spitting-cups.

2. Nurses are to see that the spitting-cups and bed-pans are properly supplied with the disinfecting solution provided—carbolic acid solution (1 in 20)—and are on no account to allow the contents of them to become dry.

3. The contents of the bed-pans are to be mixed with more disinfectant before being emptied, and the vessels are then to be thoroughly washed with boiling water.

4. In dusting the galleries special care is to be taken to wipe up the dust and not to raise it into the air. The duster should be damp and should not be shaken. All dusters after use are to be treated as soiled linen.

5. Nurses are to call the attention of the Sister-in-charge to any linen soiled by expectoration or otherwise, and such linen is to be kept separate and boiled before being sent to the laundry.

6. Nurses should be careful to wash their hands after contact with expectoration.

Directions to Patients

(*These are suspended in the Galleries.*)

1. Patients are required to use the spitting-cups provided in the hospital and grounds. Any patient infringing this rule by spitting upon the walls or floor, or in the fireplaces or washing basins, or elsewhere, is liable to be discharged.

2. Patients are required to use the pocket handkerchiefs provided by the hospital. Each patient will be provided with a clean handkerchief daily.

3. The expectoration should on no account be swallowed, as by so doing other organs of the body may become affected.

Directions to Out-Patients

(*These are placed in the Waiting Room.*)

Inasmuch as consumption is under certain circumstances a communicable disease, and as the chief source of danger is the expectoration or phlegm, *especially when it has become dry*, the following precautions are desirable :

1. Never spit about the streets or on the floor or in the fireplace, or into any vessel unless it contains a disinfectant.

2. When indoors always use a cup or jar or hand spittoon containing a tablespoonful of strong solution of washing soda or

other disinfectant. Empty it daily down the water-closet; never empty it upon the dust-heap.

3. When empty wash it thoroughly with boiling water and add a tablespoonful of the disinfectant (carbolic acid solution, 1 in 20).[1]

4. Handkerchiefs used by consumptive patients should be frequently changed, at least once daily, and should be scalded before being washed.

5. Do not swallow the expectoration, as by so doing other organs of the body may become affected.

6. Keep your room well aired and clean.

7. A consumptive patient should, if possible, occupy a separate bedroom.

8. Any room which has been occupied by a consumptive patient should be thoroughly cleansed before again being used.

9. Milk may be a source of danger, and should be boiled before use.

It has been shown that, if proper precautions are adopted, the dust from wards inhabited by tuberculous patients does not contain the virus of the disease; but, in the dwellings of the poor, rooms so occupied are very likely, owing to neglect of proper safeguards, to become centres of infection. It will doubtless in time become the general practice to thoroughly disinfect such rooms, and in the interests of the community it should, as is now the case in some towns, be done at the public expense in the case of the poorer classes.

Various portable spittoons have been devised, but it is difficult to induce patients, at any rate in this country, to carry them, and, provided the pocket handkerchief is frequently changed, and when soiled is plunged into boiling water, little risk of disseminating the virus is entailed by its use.

The possibility of the transmission of the disease from husband to wife has been already discussed (*vide* p. 306). Localised tubercular infection has been clearly proved in a few cases to have been due to kissing on the lips.

General.—The arrest of the disease is effected by the agency of forces which are inherent in all living organisms, and these are continually in operation even when the morbid process is most active.

The main object of treatment is, therefore, to develop this resisting power of the individual, and so to enable the tissues either to destroy the specific organism, or to shut off from the body areas of disease in which it may be present.

The maintenance of the nutrition is one of the most important objects to be kept in view in the treatment. At some future date the proper mode of administration of a specific remedy may occupy this place, but it is not so at present. Every patient with tuberculosis of the lungs should endeavour to increase in weight; if he can grow fat he need have little fear that the disease is making progress.

---

[1] This is given to the patients.

Hospital patients who have come from poor homes and have been ill fed may gain weight whilst the disease continues to advance, but this is rarely the case with those who have throughout their illness enjoyed every comfort which money can provide.

Although in such cases the weight may be maintained whilst the appetite continues good, there is, as a rule, a close relation between the activity of the morbid process, as evidenced by the type and degree of the pyrexia, and the state of the nutrition. Tubercular patients with active disease require a much larger quantity of food than those in health, in order to compensate for the increased waste of tissue which it entails, and if the digestion is unimpaired they can usually assimilate well.

In addition to the ordinary hours of meals, there are certain times in the day when food may with advantage be given to such patients; these are early in the morning, about 11 A.M., just before bedtime, and in some cases during the night. A glass of hot milk given before the attack of coughing, which commonly occurs shortly after waking in the morning, assists expectoration and counteracts the feeling of exhaustion which often follows, and a tablespoonful of rum may be usefully added to the milk in cases with advanced mischief. At 11 A.M. a tumblerful of milk with some biscuits, or a cup of beef tea, is preferable to the glass of port wine which is commonly recommended in such cases at this time. At bedtime a cup of some warm food prepared with milk, such as bread and milk sweetened, or arrowroot, helps to induce sleep, and by promoting expectoration relieves the attack of coughing, which in chronic cases often occurs on lying down.

Some hot liquid food during the night generally relieves cough and promotes sleep more effectually than the administration of a sedative linctus or a draught. Attention to this point is a matter of the greatest importance in the treatment of all forms of tuberculosis accompanied by active changes in the lungs. Various 'food-warmers' are sold in which the heat from a night-light is utilised for the purpose of warming liquid food.

The food taken at the ordinary mealtimes should be plain, well cooked, and as nourishing and varied as possible. Mutton and beef with fatty and starchy foods should enter largely into the dietary, but it is not, in our experience, either necessary or desirable to formulate a rigid diet table for such patients. They find it irksome, and usually discard it after a short trial. Individual proclivities require to be considered, and they will be found to vary widely.

During periods of acute illness, the diet should be abundant even to the point of excess, and such as is suitable for the febrile state. The use of various special articles of food has been from time to time advocated, but in this country, at any rate, they have not retained a permanent place in the dietary.

The 'Koumiss' treatment, as carried out in Tartary, and particularly in the region of Tsamara on the borders of the Caspian Sea, consists in drinking large quantities of the fermented milk of mares, unworked, and grazing on the Steppes. Koumiss is prepared from cow's milk and sold in this country, but its use at the Brompton Hospital has not been attended by any very marked results. The Koumiss treatment, like many others, is attended with greater success in its native home than elsewhere, but its use has proved of undoubted value in cases accompanied by irritability of the stomach and frequent vomiting. Two pints or more may be given during the day, other kinds of food being almost withheld.

The writer has no personal experience of the value of the 'raw-meat treatment.' It is highly spoken of on the Continent, but is hardly likely to come into favour here.

Alcohol.—Stout, bitter ale, wine, or alcohol in some form taken with meals is often of great service by promoting appetite, and its use may enable a tubercular patient to take a much larger quantity of food than he otherwise would. The administration of large quantities of alcohol, apart from the urgent necessity which may arise from failure of vital power, has not, however, in the writer's experience been necessary, although some authorities, notably Flint, Brehmer of Göbersdorf, and Dettweiler of Falkenstein,[1] speak strongly in its favour. There is no evidence that the admittedly injurious effects of alcohol taken apart from food are not experienced in this disease, and we have frequently observed that patients who have previously been addicted to alcoholic excess suffer, when attacked by tuberculosis, far more severely, chiefly from loss of appetite, inability to digest food, and a very irritable cough, than those who have led temperate lives. During periods of acute illness and in the later stages of a chronic case, when strength is failing, brandy or champagne is the best stimulant.

Open-air treatment.—There is a general agreement as to the great value of the mode of treatment to which the above name is now applied, which was first systematised and successfully carried out by the late Dr. Brehmer at Göbersdorf, in Silesia.

Many other Sanatoria have been founded in Germany upon his model; of these, Falkenstein in the Taunus, until lately conducted by Dr. Dettweiler; Rieboldsgruen in Saxony (Dr. Driver); St. Blasien in Baden (Dr. Haufe); Göbersdorf (Dr. Roempler); Hohenhonnef on the Rhine, Altenbrak, and St. Andreasberg in the Harz; Heidschloss (Holstein); Nordrach (Baden); Schömberg (Wurtemburg); Rehburg (near Hanover) are the chief. Similar institutions are also to be found at Davos and Arosa in Switzerland.

It will be seen that most of these Sanatoria are situated in mountainous places, although not at such high altitudes as the Swiss Alpine resorts.

In these institutions the patients live under the close supervision of the resident physicians, and every detail of their life is regulated in a manner rarely attempted in this country, and to which few Englishmen are willing to submit at the bidding of their own countrymen. The leading idea of the method is that as much of the day as possible should be spent in the open air. Either continuously or in the intervals of exercises the patients lie in covered balconies or in 'liegehalle,' or in hammocks stretched between pine trees and exposed to the sun and protected from the wind.

---

[1] Quoted, but not endorsed, by Dr. Burney Yeo, p. 98.

Patients with active or advanced disease remain at rest in bedrooms with open windows, either all the day or during the period when fever is present, whilst those who are less seriously ill spend some hours daily in walking or hill-climbing, and rest in the intervals. Most of the patients thus pass from seven to eleven hours daily in the open air in all kinds of weather. Dettweiler attaches most importance to rest in the open air, but Brehmer regards methodical hill-climbing as a factor of great importance in this method of treatment. Much importance is rightly attached in these Sanatoria to the maintenance of nutrition, food being taken frequently but in moderate quantity at each meal. The following is the dietary and distribution of mealtimes generally adopted:[1]

'Breakfast between 7 and 8 A.M., consisting of coffee, cocoa, tea, white or brown bread and butter, and a glass of milk. At 10 o'clock, luncheon, consisting of from one to two glasses of milk and bread and butter, or perhaps broth, eggs, &c., and a glass of wine to finish with. At one o'clock, dinner of soup and three other courses, two of meat with vegetables and one of pudding, with one or two glasses of wine. About 4 P.M. afternoon tea (a repetition of the early breakfast); at 7 P.M. supper, of one or two courses, one cold and one hot with vegetables, and one glass of wine; at 9 P.M. a glass of milk with two or three teaspoonfuls of cognac.'

The above applies to ordinary cases, not to such as from the presence of fever or other cause are upon a special diet. The following results are claimed by Dr. Brehmer of Göbersdorf to have followed this mode of treatment in 5,032 cases out of 5,440 during the eleven years from 1876–1886. No detailed reports are available of the remaining 408 cases.

Stage of disease	Number	Cured	Nearly cured	Total
I.	1,390 (27·6%)	387 (27·8%)	430 (31%)	817 (58·8%)
II.	2,225 (44·2%)	152 (6·8%)	325 (14·6%)	477 (21·4%)
III.	1,517 (28·17%)	12 (·8%)	33 (2·3%)	45 (3·14%)
—	5,032	551 (11%)	788 (15·6%)	1,539 (26·6%)

---

[1] *Sanatoria for Consumptives*, Von Jaruntowsky. English translation by Dr. E. Clifford Beale.

Inquiry made in 1890 into the subsequent history of cases described as 'cured' or 'nearly cured' showed that in five cases the cure had lasted from 20 to 29 years, in 52 cases from 12 to 21 years, in 38 cases from 7 to 12 years.

Of forty patients discharged in 1876 as cured or nearly cured, and of whom particulars could be ascertained in 1890, twenty-five were then living and in good health, one was suffering from 'fibroid phthisis,' one died in 1886 from 'phthisis,' and thirteen others had died from unknown causes.

For the following account of the Sanatorium at Nordrach, which is under the direction of Dr. Otto Walther and can be thoroughly recommended, the writer is indebted to a former House Physician at the Brompton Hospital, himself a sufferer from tuberculosis and for twelve months a patient under treatment at Nordrach.

We take this opportunity of stating that we have never observed, coincident with the complete arrest of advanced disease, a more complete physical change in any individual. Before the onset of the disease he was thin and somewhat gaunt in appearance; his height was 5 ft. 10 in., and his weight 11 st. After a severe illness, marked for fifteen weeks by continuous pyrexia and great emaciation, he went to Nordrach, whence he has lately returned so altered as to be scarcely recognisable, presenting an appearance of robust health, somewhat stout, and weighing 13 st. 10 lb.

Physical examination of the chest indicates that complete arrest of the disease had taken place.

'Nordrach is a small village in the Baden Black Forest, nine miles from Biberach Zell, the nearest station on the Schwarzwald Bahn, and thirty miles distant from Strasburg. It is situated in a cul-de-sac at the remote end of a beautiful sub-alpine valley, 1,400 ft. above the sea. The valley is open to the south-west, and shut in by a horse-shoe-shaped range of hills rising another 1,400 ft. on the north and east. These hills are clothed with miles of pine forests, which prevent dust and afford walks sheltered from wind. The rainfall of the district is considerable at all seasons, so that the air is frequently washed free from impurities, and the floor of the valley is kept green by many springs and streams. On the other hand the geological formation is red sandstone, which rapidly absorbs the heaviest showers and leaves all paths in good condition for walking. The climate is almost as variable as that of England, but warmer in summer and colder in winter. The atmosphere is exceedingly pure, free from dust and charged with moisture, which has a marked influence in allaying cough. The Sanatorium consists of four houses, containing in all about forty-two bedrooms and a separate dining-hall, all carefully constructed to avoid the formation of dust, to cause patients while indoors to be practically in the open air, and to give the minimum exertion to those in bed.

'To these ends the walls and ceiling of each room are of varnished wood, the floor covered with linoleum, which is swabbed with water every morning; there is ample window space, and a hot-air heating apparatus. The large casement windows are practically open all the year round, so that there is the least possible difference between the temperature of the air within and without; as a consequence, colds are almost unknown. The walks on the surrounding forest slopes are extremely varied and picturesque, the nearer specially laid out on easy gradients with benches at short intervals, and there are several small shelters for storms; and the more distant paths are everywhere open to patients. Of amusements, with the exception of occasional music, there are practically none, excitement and irregular exertion of all kinds being regarded as hindrances to the absolute mental and physical quietude which is considered a necessary part of the treatment. The main features of the treatment by which it is sought to effect a really permanent cure are as follows:

'1. An absolutely open-air life for every variety of case, whether acute or chronic, accompanied by fever or apyrexial, in all weathers and seasons, by night as by day.

'2. A regular course from first to last of over-feeding—stuffing would be a more appropriate term—with a rich and varied diet, including much meat, milk, fatty and farinaceous food, given in large quantities at a time, with long intervals between the meals.

'3. A judicious combination of exercise (hill-climbing), carefully regulated and carried out so as always to fall short of producing either dyspnœa or fatigue, in order not to interfere with the processes of repair, with the maximum amount of mental and physical rest.

'4. Every patient is under the constant personal supervision of the physician—a vital and distinctive factor in the treatment.

'During a year's residence at Nordrach the writer has never known a patient take a cold or chill. Every arrangement tends to force patients into the open air, and to overcome their natural timidity of draughts and damp clothes. It is found from experience, which has been very fully tested, that no amount of exposure to draughts or wet, or any variation of weather, causes the most delicate patient to take cold or any other harm, so long as an open-air life is led. Casement windows forming almost one side of the bedroom are almost always open, though closed at times in winter to warm the room when it is unoccupied. The windows of the dining-hall are actually taken out during a large part of the year. Patients have no sitting-rooms, and must be out all morning for the daily walk, however short. This kind of life, at first uncomfortable, becomes in time quite natural, induces a hardening to cold and heat, and renders a return to town life at first quite distasteful.

'Three meals are given in the day; breakfast, at 8 A.M., consists of coffee, bread and butter, and cold meat, such as ham, tongue, sausage, &c., and a half litre of milk. This after a time is reduced to a quarter litre, according to the patient's capacity and need for putting on flesh. Dinner at one o'clock consists of two hot courses of meat, or fish and meat, about four to six ounces being served to each patient, with plenty of potatoes and green vegetables, and sauces in which butter is the main ingredient. The third course may be pastry, or farinaceous pudding, fruit and ice cream, with coffee and a half litre of milk. Supper at seven usually consists of one hot meat course as at dinner, and one cold, as at breakfast; tea, and a half litre of milk. The two latter meals must be taken under the supervision of the Resident Physician, and servants may not take away the plates until everything has been eaten. Roughly speaking, the patient eats about double the amount of food he desires. Pyrexial cases in bed have the same diet, with if anything slightly larger portions; and it is found that absolute rest in bed and over-feeding are the most potent means of reducing temperature. Patients rest on their couches from 12 to 1, and from 6 to 7, before meals. On this plan, patients invariably gain weight, averaging two, three, and even four pounds a week for the first few weeks, and afterwards by smaller increments. Not only fat but firm muscle is formed, and it is not uncommon for patients to gain one quarter to one third of their total weight in this way.

'Patients are not unfrequently sick during or shortly after a meal; if this happens, they come back and finish the meal, finding as a rule no difficulty in retaining the remainder.

'The rest before meals and the long intervals between are valuable aids to complete assimilation, and dyspepsia is on the whole rare; if present, it usually disappears as nutrition improves.

'Cases of active disease with high fever are treated by rest in bed, over-feeding, and usually by isolation, any exertion, such as talking, being spared as much as possible. When the temperature remains below 38° C. in the evening, and below 37° C. in the morning for a week or ten days, the patient is allowed a short walk in the morning, beginning with fifty yards or so, and gradually increasing the distance tentatively. If the temperature tends to rise in the mornings to 37° C. or above, rest is resorted to again.

'Patients take their own temperatures in the rectum four times daily: on waking, at 12 M., immediately after the walk, at 5.30 P.M., and before going to bed. An average good morning temperature would be 36·3 C. ·4, or ·5, and after the walk a degree or a little more higher. This exertion temperature should in most cases not exceed 38° C. and must be taken at once, as it often falls rapidly in the first few minutes.

'Walking must be at a very slow uniform pace, and at first several rests should be taken, so as never to cause the least fatigue. Talking, except during rests, should be avoided, particularly when going uphill, and special cases are sometimes sent to walk by themselves. The first part of the walk is usually an ascent, then a horizontal portion and a descent home.

'As energy revives, the distance is cautiously increased, regard being given to the temperature, until several miles can be done, involving a climb of five or six hundred feet during the morning; but a slow, steady gait must always be observed. The afternoon is usually spent in rest or a short walk, and patients go to bed about nine to half-past. The physician visits each patient three times daily, before each meal, except those who are convalescent; to such patients one visit daily is considered sufficient.

'Every patient has one of Dettweiler's pocket flasks, and a cup in his bedroom to receive sputum, and spitting elsewhere is forbidden.

'The chest is examined monthly, and at the same time the expectoration is very carefully examined for tubercle bacilli. The results of this treatment are in the majority of cases extraordinary. Dyspepsia disappears, anæmia gives place to a healthy colour, weight is made to the extent of twenty to fifty pounds, cough and expectoration diminish, and physical signs correspondingly decrease, the whole constitution becoming totally reorganised. Finally, bacilli can no longer be found in the expectoration. As a final test, some expectoration is injected under the skin of a guinea-pig and an interval of three or four weeks is allowed to elapse. If no signs of tuberculosis develop in the animal, the patient is allowed to return home, often to do moderate work at once, which is regarded as the best mode of securing the favourable changes induced in the lungs.'

We are glad to hear that the writer proposes to establish a sanatorium conducted on similar lines in this country, as such an institution is much needed, and England is now far behind continental countries, particularly Germany and Switzerland, in the facilities it affords for the 'open-air' treatment of tubercular disease of the lungs.

Exercise.—The more exercise the patient is able to take in the open air the better; the exact form must depend upon a variety of considerations, but in all cases it should stop short of producing fatigue. Riding on horseback, or, for moderate distances, on a bicycle, although tiring to the beginner, are modes of taking exercise which entail comparatively little effort upon the experienced. Walking, particularly up gentle ascents, is especially valuable as promoting more complete expansion of the lungs.

For patients with limited, or more or less completely arrested, disease, many of whom are found during the winter at St. Moritz, Davos, and other Alpine resorts, skating is, whilst they are there, a most suitable exercise. Curling and golf are also good forms of exercise for such patients. When no longer strong enough for these forms of exercise already mentioned, it is generally advisable for the patient, if his means allow of it, unless the disease is very advanced or there is high and continuous fever, to be out in a carriage or a bath chair, or reclining in the open air in some protected spot during part of the day.

Specific treatment: tuberculin.—On August 4, 1890, eight years after his memorable discovery of the tubercle bacillus, Koch stated, at a general meeting of the International Medical Congress at Berlin, that he had, as a result of prolonged research, found 'a substance which, when inoculated into a guinea-pig, renders it incapable of reacting to the inoculation of the tubercular virus; the same substance in guinea-pigs already attacked with advanced and generalised tuberculosis brings about a complete arrest of the development of the malady without the animal suffering any harmful effects from the influence of the remedy.'

On November 13, 1890, Koch published[1] the results observed to follow the use of this remedy in tuberculous patients.

The substance proved to be a glycerine extract of pure cultures of the tubercle bacillus.

The great interest excited throughout the world by this announcement is still fresh in our memories; the lamentable lack of judgment, of self-control, and of those critical faculties which should characterise the members of a scientific profession, displayed by many upon that occasion, may serve as a warning for the future. The history of medicine in still more recent times, however, appears to prove that the tendency to exploit for selfish purposes each new discovery in therapeutics, and to recommend it as a panacea for almost every ailment of mankind, is ineradicable from the minds of the weaker brethren.

The results of a prolonged and careful trial of tuberculin in cases of pulmonary tuberculosis under the care of eight physicians at the Brompton Hospital are thus summarised in a report signed by Drs. C. Theodore Williams and John Tatham:

'1. That tuberculin, if introduced under the skin, speedily causes inflammatory changes in and around tubercular lesions.

'2. That the action of tuberculin in lung tuberculosis is to cause breaking down of the tubercular masses and of the lung tissue in the neighbourhood, and thus to promote the formation of cavities. That this is the case is proved by (*a*) the appearance of lung tissue in the sputum, where it was previously absent, and (*b*) by the physical signs of cavity replacing those of consolidation.

'3. That tuberculin increases the amount of expectoration, but that there is no proof that it diminishes the number of tubercle bacilli contained therein, for, in some of the patients, they apparently increased under its use.

'4. That in many cases tuberculin injections are followed by a distinct extension of disease, as evidenced by physical signs.

'5. That the reactions due to tuberculin are exhausting to the patient, and cause loss of weight and strength.

'6. That this treatment is specially contra-indicated in lung tuberculosis accompanied by pyrexia, as likely to convert intermittent into continuous pyrexia.

'7. That lung excavation accompanying the use of tuberculin may be followed by contractile changes, due to increase of fibrosis. This was shown in two of the cases under observation, where diminution of cough and expectoration and gain of weight took place.

'8. That the tuberculin did not favourably influence the course of the disease in the majority of cases; that in some the effects were detrimental; and that even in the stationary and improved cases it was difficult to ascribe any distinct improvement to the injections, which might not have been equally attained under the treatment ordinarily employed in the hospital.'

---

[1] *Deutsche Med. Wochenschr.*, No. 46*a*.

Professor Koch has recently[1] stated that he still regards tuberculin as the best remedy against tuberculosis, but at the same time he brings forward three new preparations—viz., tuberculin A, O, and R, of which the last is recommended for clinical use.

'Tuberculin O and R are prepared by vigorously pounding in a mortar dried cultures of the tubercle bacillus, and then adding distilled water. The whole is centrifugalised in a powerful machine. The clear but opalescent liquid collecting at the top contains no tubercle bacilli, and constitutes in the first centrifugalisation tuberculin O. The remainder, containing the *débris* of the tubercle bacilli, forms tuberculin R. This remainder is dried and pounded, distilled water added, and the whole again centrifugalised. This process is repeated until hardly any residue remains. Twenty per cent. glycerine is then added to both preparations to ensure their preservation. The dose, by subcutaneous injection, is 1–500 mgr. to begin with. An injection is given every other day, and the dose is gradually increased up to 20 mgr.'

If the temperature rise the injections should be stopped.

It is claimed for the tuberculin R that it exerts both immunising and curative effects, and that cases of tuberculosis and lupus in an early stage are without exception greatly improved by its administration, and that no unfavourable results attend its use.

Guinea-pigs thus treated were so completely immunised that injections of virulent cultures had no effect.

Our experience of this remedy is not yet sufficient to justify any statement of opinion as to its value.

Koch states that cases of tubercular disease in which the temperature rises above 100°·4 F. are not suitable for this mode of treatment, as such a degree of fever indicates the presence of mixed infection (streptococci and septic organisms). We have considered this question elsewhere (*vide* p. 324).

The discovery of tuberculin has undoubtedly proved of the greatest service to medical science, both by suggesting work on similar lines in connection with the toxines of other diseases, and by providing a test for the presence of tubercle in cattle. When its use for this purpose becomes general, and the slaughter of animals thus proved to be infected is made compulsory, milk will no longer be a source of infection, and the incidence of tubercular disease in childhood will probably be markedly diminished. Its value as a test for the presence of tubercle in human beings is not great, as reactions are obtained in several other diseases.

A chart of the temperature during the 'reaction' produced by the injection of tuberculin is subjoined in order to emphasise the fact that it is a remedy which produces fever.

---

[1] *Deut. Med. Woch.*, April 1, 1897, p. 209.

We are strongly of opinion that no remedy which has this effect is admissible in the treatment of tubercular disease of the lungs.

The patient from whom this chart was obtained was under treatment with tuberculin for a period of thirty-six days, during which thirty-one injections of tuberculin were given. The dose was gradually increased from ·001 cc. to ·1 cc. During the course of the treatment four calcareous particles, one of which was surrounded by pigmented lung tissue and also another small portion of lung tissue, were expectorated, and signs of a small excavation appeared at the apex of one lung.

These facts are sufficient to prove the great danger of lighting up active changes in quiescent lesions which attends the use of tuberculin in doses sufficient to produce fever. Fortunately the form of disease present in this case was markedly fibroid, and no subsequent ill-effects followed the treatment adopted.

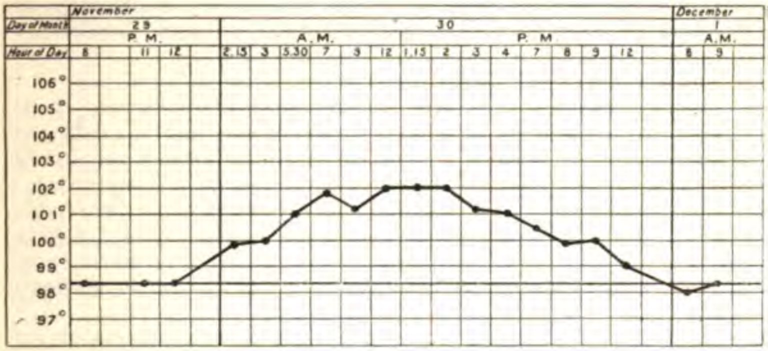


FIG. 109.—PYREXIA DURING THE 'REACTION' PRODUCED BY THE INJECTION OF TUBERCULIN

Klein (Arthur)[1] has lately attempted to prove that the action of tuberculin is not upon the specific virus of the disease, but upon the pyogenic organisms, streptococcus pyogenes, pneumococcus and the staphylococcus aureus, present in association with the tubercular lesions, and that the phenomena of the 'reaction' are due to the multiplication and increased virulence of these organisms. He was led to this conclusion primarily by the discovery of inflammatory lesions in which these organisms were present in great abundance around tubercular foci in patients dying whilst under treatment with tuberculin. This view, however, is disproved by the fact that the reaction is observed in perfectly healthy animals, and also by the clinical observation that with fibroid pulmonary lesions the

---

[1] *Ursachen der Tuberculinwerkung*, 1893, Wien.

For internal administration we prefer pure beechwood creasote, which is best given in the form of '*perles*' or palatinoids, each containing two or three minims of the drug. Of these perles two may be given three times daily with meals to begin with, and the dose may be gradually increased until the patient is taking from fifteen to twenty minims of the drug daily. Some writers, however, advocate the use of much larger doses. We are glad to observe that a protest has recently been raised against the use of this drug in the excessive doses sometimes recommended, a method of treatment to which the term 'creasotic orgie' (!) has been applied. We have rarely found creasote interfere with digestion; if this should happen, its use should be discontinued, as in the treatment of tuberculosis the assimilation of food is of far greater importance than the administration of any drug.

Guaiacol carbonate may be given in cachets, in doses of eight to fifteen grains three times daily, and the dose may be gradually increased to twenty grains or even more. Benzosol may also be given, either in cachets or tabloids, in similar doses.

Sterilised oil of guaiacol may be administered by subcutaneous injection, but no decided advantage counterbalances the obvious objections to this mode of treatment.

Of the direct injection into the lungs of creasote or guaiacol we have no experience, and the use of these remedies in this disease by the method of intra-laryngeal injection has not been in our experience attended by any benefit.

Various other remedies of the antiseptic class have from time to time been employed, but as we have no experience of their use we refrain from mentioning them. For these, as for so many other remedies, the favourable results at first claimed have not been substantiated by subsequent experience.

So soon as any remedy is discovered which exercises a decided influence upon the virus, its effects will probably be so easy to recognise that the matter will be speedily placed beyond the possibility of doubt.

Antiseptic agents may also be employed in the volatile form as inhalations or sprays. Dr. Burney Yeo speaks highly of the favourable effects of the continuous inhalation of equal parts of either creasote or carbolic acid and spirits of chloroform, in modifying the course of the disease and preventing infection of the larynx; and Wilson Fox admitted the value of this method of treatment. One of the chief results observed to follow its use is a diminution in the amount of the expectoration.

Eucalyptol, iodine, iodoform in ether, and oleum pini sylvestris are other agents of this class which may be used in the form of inhalations.

Tonic remedies.—Of these the most valuable are cod-liver oil, the hypophosphites, arsenic, quinine, nux vomica, mineral acids, and vegetable bitters such as gentian. As a general rule, their usefulness is most marked dur[illegible]ods of quiescence of the disease.

Cod-liver oil.—Owing to improved methods of preparation, cod-liver oil is now far less nauseous than formerly, and only the pure pale 'tasteless' oil should be prescribed.

The favourable effects of this remedy are specially observed during the apyrexial periods of the chronic variety of the disease, a steady improvement in the nutrition of the body being the best evidence of its beneficial action.

Continuous fever or even a high evening temperature, and the presence of gastric or intestinal catarrh, are conditions which contra-indicate its use, and it is rarely wise to force the remedy upon a patient who expresses a decided distaste for it, and never if it produces nausea and impairs the appetite. As a rule, patients take the oil much more readily during cold than hot weather; many who can assimilate it during the winter months being obliged to discontinue its use during the height of summer. Children usually take cod-liver oil without difficulty, and many acquire a decided taste for it.

The best effects are obtained from moderate doses; one to two teaspoonfuls may be taken twice or three times daily after meals for long periods. Some patients, who are unable to digest the oil after meals, are able to take it at bedtime without subsequent nausea.

Cod-liver oil may be enclosed in capsules, or its taste may be disguised by some vehicle, such as orange or steel wine or lemon juice. At the Brompton Hospital, cold coffee with salt sprinkled on the surface of the oil has been found efficacious for this purpose. Some, who are unable to digest it when pure, can take it in the form of an emulsion, or in combination with creasote (creasote, ♏v—x, ol. morrh. ʒij, twice daily).

Many remedies, such as pancreatic emulsion, cream, maltine, are used either as substitutes for cod-liver oil when from one cause or another the oil cannot be taken, or are given in combination with it. All those named are of service; glycerine is another, but of more doubtful value.

The administration of the hypophosphites of calcium, sodium, and potassium, but especially the former, is indicated in the early and less acute stages of chronic tuberculosis, and particularly in young subjects. These remedies may be given in the form of syrups, and in combination with iron and other tonics. The following is a useful formula: ℞ calcis hypophosphitis, gr. v.; syrupi ferri phosphatis, ʒj; glycerini, ʒj; inf. gent. co. ad ℥j; ter in die.

Arsenic.—In the early periods, and in chronic cases combined with asthma, much benefit has been observed to follow the administration of arsenic (Williams). Many physicians indeed regard it as a remedy of the greatest importance. Our own experience, however, is in accord with that of Dr. Burney Yeo, who states that he has not obtained any very brilliant results from its use. It is chiefly of service in cases associated with anæmia.

Iron, in the form of the syrup of the iodide or phosphate, is

A similar method of treatment is often efficacious in relieving cough which occurs on lying down at night.

If the cough continues during the night and prevents sleep, a dose of a linctus containing codeina or morphia should be given, and if necessary it may be repeated; but the less often it is taken the better.

Codeina (gr. $\frac{1}{6}$), citric acid (gr. vj), spirits of chloroform (♏iij to v), and a drachm of mucilage of acacia, or liquor morphinæ acetatis (♏viij), chloric æther (♏iij), lemon-juice (♏xv), and a drachm of mucilage of acacia, are useful combinations. The use of sedative linctuses during the daytime should be avoided as much as possible.

An irritating and ineffective cough, persisting during the day, is best treated by the use of inhalations or sprays.

Ten drops of a twenty per cent. solution of menthol in alcohol may be sprinkled on the sponge of a perforated zinc inhaler, and this may be worn for an hour or more at a time.

Antiseptic drugs, such as creasote, carbolic acid, eucalyptol, or the oleum pini sylvestris, in combination with a sedative volatile agent such as chloroform, may be used in a similar manner, or they may be employed in the form of sprays.

In addition to relieving cough, these remedies are of service by diminishing the quantity of the expectoration.

Counter-irritation by the use of blisters or liniments containing turpentine and iodine (*e.g.* linimentum terebinthinæ, ʒvj; tinctura iodi, ʒj), or a pigment composed of equal parts of the tincture and liniment of iodine, is often efficacious in relieving cough.

Cough arising from laryngeal irritation may be relieved by the inhalation of medicated vapour. For this purpose a variety of remedies, such as conium, henbane, hop, or tincture of benzoin may be employed. The following is a useful formula: ℞ chloroformi, ♏x; succi conii, ʒj; glycerini acidi carbolici ad ʒij, to be added to half a pint of boiling water and the vapour inhaled.

Pain referred to the axillary regions, and possibly due to a localised pleurisy, may generally be relieved by strapping the side. Pain over the front of the chest is best treated by the application of iodine, or by the use of a liniment of belladonna. The effect of iodine, however, requires to be carefully watched, as the sensitiveness of the skin to its action varies greatly in different individuals, and in some persons intense irritation (which is apt to be mental as well as local) soon follows its use.

Night sweating.—In the majority of cases this symptom is associated with pyrexia, the sweating occurring regularly in the early morning when the temperature begins to decline. Relief may often be obtained by sponging the body with cold or tepid water at bedtime in the manner recommended in the treatment of pyrexia. Quinine in combination with digitalis often acts well in such cases. If these should fail, a trial may be given to oxide of zinc (gr. iij–iv), either alone or in combination with extract of belladonna (gr. $\frac{1}{3}$–$\frac{1}{2}$), administered in the form of a pill at bedtime, or to atropine in doses of $\frac{1}{100}$ to $\frac{1}{60}$ gr. in pill, or to sulphuric acid. The disadvantage attending the use of the various preparations of belladonna is their tendency to produce dryness of the throat. Arseniate of iron (gr. $\frac{1}{6}$–$\frac{1}{3}$), nitrate of pilocarpine (gr. $\frac{1}{20}$), picrotoxine (gr. $\frac{1}{60}$), agaricin (gr. $\frac{1}{13}$), have all been found of service in cases which have not yielded to the remedies mentioned above.

When profuse sweating invariably attends sleep, it is generally a sign of exhaustion, and may be prevented by the administration of stimulants and hot liquid food at bedtime or in the early morning. When the patient wakes in a profuse perspiration, the clothing and bed-linen should be changed at once, the skin thoroughly dried, and some hot milk and brandy given. If the sweating, as is not infrequently the case, follows a prolonged paroxysm of cough, the chief attention should be given to the treatment of the latter symptom.

Diarrhœa.—In cases of acute ulceration of the intestine, rest in bed, the application of hot linseed-meal poultices to the abdomen, and the restriction of the diet to milk, meat essences, jelly beef-tea, and chicken broth, given at intervals of from two to three hours, constitute the most important measures of treatment. A small dose (ʒj–ʒij) of castor oil should first be given to clear the bowel of any contents which may be a cause of irritation, and subsequently subnitrate of bismuth in doses of gr. x–xv combined with opium (gr. $\frac{1}{4}$), administered in powders every four hours.

Diarrhœa due to chronic ulceration of the intestine occurring in the later stages of the chronic form of the disease is usually best treated by restriction of the diet to such articles as are easily absorbed and leave little residue, and by the administration of astringents combined with opium. Subnitrate of bismuth (gr. xx–xxx) with pulv. ipecac. co. (gr. x) forms a useful combination, which may be given in milk every four hours.

Mineral astringents such as acetate of lead (gr. ij–iij) and sulphate of copper (gr. $\frac{1}{4}$–$\frac{1}{2}$), in combination with opium, are also of use when the remedies already mentioned fail.

Kino, catechu, and hæmatoxylum may also be tried in combination with opium (tinct. catechu ʒss; tinct. opii ♏v–x; mist. cretæ ℥j).

The fluid extract of coto bark in doses of five minims, combined with compound tincture of cardamoms and mucilage is an efficacious remedy in advanced cases.

An enema of starch and opium (tinct. opii ʒss; mucilag. amyli ℥ij) is often of great service when the large intestine is the part of the bowel chiefly affected. Its use may be combined with the administration of a pill of acetate of lead and opium.

If catarrh of the intestine is the cause of the diarrhœa, after the bowels have been cleared of any irritant by the use of a mild aperient, such as castor oil, the carbonate or subnitrate of bismuth may be given. Salol, salicylate of bismuth, and creasote may be used when diarrhœa is accompanied by marked fœtor of the evacuations (*e.g.* salol, bismuthi salicylatis, sodii bicarb. āā gr. v in cachet).

Disorders of digestion.—We have already dwelt upon the fact that the resisting power of the patient mainly depends upon the condition of his digestive organs. It is apt to be overlooked that the ingestion and assimilation of food are not the same thing, and that nutrition depends upon the latter only; if, therefore, the disorder is due to the quantity of the food taken being in excess of the digestive power, it must be diminished. The best evidence of this condition is the presence of undigested food in the stools.

Loss of appetite, a pale tongue, and a sense of weight after meals are indications for the administration, shortly after food, of mineral acids with strychnia or some other vegetable bitter. Pepsine and hydrochloric acid often aid the digestion of food in such cases.

Loss of appetite without digestive disorder is usually best treated by the administration of alkalies with vegetable bitters shortly before meals. We know of no combination so efficient as the mistura gentianæ alkalina of the Brompton Pharmacopœia, which contains bicarbonate of soda (gr. xv), dilute hydrocyanic acid (♏iij), and compound infusion of gentian (℥j).

Gastric catarrh, with a furred tongue and flatulent distension of the stomach after food, is often relieved by hot water taken in the early morning, and by the administration of carbonate of bismuth with hydrocyanic acid. Creasote is also useful under the same conditions. Attention to the diet and to the condition of the bowels is specially necessary in such cases.

Pain in the region of the stomach and vomiting may be relieved by the administration of bismuth and lime-water, or of an effervescing mixture containing morphia.

Climatic treatment.—The knowledge that is necessary in order to form a trustworthy opinion as to the place best adapted for a particular sufferer from this disease can rarely be obtained except by a personal visit to the place, and, if the medical adviser has not this experience, he will generally find it to the interest both of the patient and himself to leave the decision in other hands.

Opinion is practically unanimous that in suitable cases the benefit resulting from change of climate—and in this statement we include the form of treatment described as the 'open-air' method—far exceeds that to be obtained in any other way. But for the favourable changes thus induced to be of permanent or even of prolonged duration, it is essential that the patient should remain for a considerable time under the influence of the conditions by the aid of which his health has been restored.

It not infrequently happens that, after a winter spent at one of the resorts in the high altitudes, the complete return of strength and the absence of morbid signs in the lungs lead both the patient and his medical adviser to think that work may be resumed. The arrest of the disease appears to be complete, as indeed it may be, but the probability of it remaining so is infinitely greater if a further period of twelve months is passed under conditions as nearly similar as it is possible to obtain.

A favourite aphorism of the late Sir George Humphry was 'nothing ever gets quite well.' It may not be absolutely true, but the sufferer from tuberculosis will do well to bear the saying in mind. When told that the disease has undergone complete arrest, or that he is 'cured' and only a 'scar' remains, he is very apt to think that no further danger is to be feared, and to govern himself accordingly. It is, however, impossible from a physical examination of the chest to state the exact pathological conditions present throughout an arrested lesion. In one case fibrosis may be complete, whilst in another life may depend upon the integrity of a fibrous capsule surrounding a caseous mass, a condition replete with possibilities of evil in the future.

All who have had much experience in the treatment of this disease must be able to recall cases, apparently of the most favourable character, in which the patient returned to work after too short an interval of rest, with the result that the mischief again became active.

The chief points to be considered in advising a patient as to a change of climate are his means and social position, his general condition so far as strength and nutrition are concerned, and the stage and degree of activity of the morbid process.

Some might be disposed to reverse this order, but in practice the advice to be given more often depends upon the ability of the patient to bear the expense which is necessarily entailed by travel or absence from work for a very long period than upon any other consideration.

In some cases, however, either the activity or the extent of the disease absolutely contra-indicates climatic treatment abroad.

Patients with caseous tuberculosis of the broncho-pneumonic type, in which continuous pyrexia is associated with extensive caseation or rapid breaking down of the lung, should never be sent abroad. Cases of the chronic type, in which acute miliary infiltration of the lung is in progress, also cases complicated with acute pleurisy or acute bronchitis, are equally unsuitable for this mode of treatment, as are also cases of laryngeal tuberculosis associated with advanced pulmonary lesions, and those in which extensive ulceration of the intestine is present.

On the border line are cases of chronic tuberculosis, in which there is extensive disease of both lungs, and all hope of its arrest has been abandoned. In such cases the inclination of the patient, and to a less degree that of his friends, should have some weight. If he or his friends dread the idea that he may end his days away from the comfortable surroundings of home, and such an event appears not only possible but probable, it is very unwise to permit him to undertake a long journey, and positively cruel to advise such a step. If, on the other hand, the patient is anxious to go, is accustomed to travel, and is buoyed up with the hope that change of climate may lead to the restoration of his health, it may be better to fall in with his wishes, as the significance of a refusal to do so is fully appreciated.

Patients with advanced disease should, however, only be allowed to travel to places at a moderate distance, within reach of their friends, and where good nursing and some at least of the comforts of a home are assured to them. A sea voyage in such cases is rarely, if ever, permissible.

Treatment at high altitudes.—The evidence in favour of the beneficial effects of a prolonged stay in the high altitudes, and particularly at St. Moritz (6,100 feet), Davos (5,300 feet), and Arosa (6,209 feet) in the Swiss Alps, is so strong that it is unnecessary at the present time to regard the question as needing discussion.

These effects are due to the purity of the air, and particularly to the absence of organic germs, to the stimulating effect of brilliant sunshine, to the dryness of the air, and to its rarefaction, which necessitates deeper inspiratory efforts, and thereby increases the functional activity of the lungs. Other factors of importance are the diathermancy of the atmosphere, which implies a low air temperature, the comparative absence of winds, and the presence in the air of a large amount of ozone.

Cases specially suitable for the Alpine treatment are those of the fibroid type and those of the chronic variety, in which the area of disease is limited and the patient was previously vigorous, but has been infected whilst run down in health. For example, after recovery from a primary attack of hæmoptysis, no signs of active mischief being present, a young and fairly vigorous patient may with confidence be recommended to betake himself to the Alps, and may, as the result of a residence there, fairly hope to obtain complete arrest of the disease.

When the disease has extended from the apex primarily affected to the lower lobe on the same side, and the other lung is also involved, if the strength and nutrition are fairly good and fever is absent or moderate, the case may still be sent to the high altitudes, and pathological experience proves that, with a limited area of disease in these sites, complete arrest is still possible.

When a cavity is present, the case is not necessarily unsuitable for the high altitudes; but a careful consideration must be given to all its features—*e.g.* the degree of activity of the morbid process, the bodily strength of the patient, and his ability to withstand cold. Dr. Hermann Weber[1] is of opinion that even in cases in which the disease has advanced to this stage, 'greater benefit may be anticipated from a prolonged or indeed an indefinite sojourn in the Alps than from any other form of climatic treatment.' And with this opinion we are completely in accord.

Cases of tubercular pleurisy, with infiltration of the lung in an early stage, are usually greatly benefited by a residence at one of the high altitude stations.

As a rule the patients who obtain most benefit from the change are young subjects who are fond of outdoor exercises, such as skating, and are strong enough to take part in them, who whilst in the mountains spend the greater part of the day in the open air, and who are determined to do all in their power to regain their health.

---

[1] Art. 'Climate in the Treatment of Disease,' Allbutt's *System*, vol. i. p. 286.

The conditions which contra-indicate a resort to a high altitude are :

(1) The presence of certain complications such as albuminuria, advanced disease of the larynx, valvular disease of the heart, and extensive emphysema.
(2) Extreme emaciation.
(3) An irritable state of the nervous system with constantly quickened pulse, and a liability to feverish attacks.
(4) A decided inability to bear cold.
(5) To these must necessarily be added those conditions which, as already stated, render a case unsuitable for climatic treatment abroad.

It is generally advisable for patients to arrive at the Alpine stations either at the end of September or the beginning of October, but many do not do so until a month later. If the disease does not declare itself until a later period of the year, it is rarely wise for a patient to start from this country after the end of January. The winter season ends at St. Moritz when the snow melts, in March or April ; patients can remain at Davos throughout the year.

The chief winter resorts in Switzerland have already been mentioned ; others are Les Avants (3,200 feet) above Montreux, Wiesen (4,771 feet), Thusis (2,448 feet) Grindelwald, Andermatt (4,738 feet), Meran (1,051 feet), Muhlen (4,793 feet), Seewis (2,985 feet), Leysin (4,757 feet), Ragatz (1,709 feet). Some of these are utilised for continuous residence during the winter, others as stopping stations in the spring by patients who have spent the winter at a higher elevation and are making a gradual descent to a lower level, preparatory to returning home. Those who return to pass the summer in the Alps usually desire a change, and may then stay at Pontresina, Maloja, Wiesen, or elsewhere.

Some patients find after a time that they are only able to live in comfort at a high altitude, and are perforce compelled to reside there continuously.

Wherever the patient may spend the winter, he will do well to place himself under the care of a resident physician, and to consult him as to the time of leaving and where it is best to break the return journey. We do not, however, consider it essential that all patients should, on leaving the high altitudes, remain for a time at some place at a rather lower level, as we have known many who have returned direct to England without suffering any harm, and we have known cases in which some of the benefit obtained at a high elevation was lost during this gradual descent in the spring, when the weather at the lower levels may be very unfavourable.

The shorter journey naturally renders the Swiss resorts more attractive to patients from this country, but there are many more distant stations at even higher altitudes which are suitable for similar cases.

In America among the chief health resorts at high elevations are Manitou (6,200 feet) and Colorado Springs (6,000 feet) in the Rocky Mountains, and Denver (5,200 feet), the capital of the State of Colorado. At some of the Colorado resorts, *e.g.* Denver, it is possible for a patient who is strong enough to work to earn a livelihood, an advantage which is not presented by the Alpine stations. Dr. Osler states that he has seen better results in cases treated in the Adirondacks than elsewhere in America.

For full information as to the various stations in Colorado, the reader is referred to Dr. Williams' work on Aerotherapeutics.

The high table-lands of South Africa in the central and upper Karroo districts are suitable in the same class of cases as the Alpine stations; but, owing to the absence of good hotels and to the rougher aspect of life, they are not visited by well-to-do English patients so much as the Swiss resorts.

The most frequented places are Tarkastadt, Cradock, Beaufort West, Kimberley, Aliwal North, Bloemfontein, Lemoenfontein, and Grahamstown.

South Africa is specially suitable for fairly vigorous young men in an early stage of the disease who know some trade, but it is essential that they should have sufficient means to enable them to live in comfort without the necessity of earning their livelihood until complete arrest of the disease has taken place, and for at least twelve months subsequently.

The chief resorts in the Andes are Santa Fè de Bogota in New Granada, Quito in Ecuador, Arequipa in Tarma, Jauja and Huancayo in Peru, and La Paz in Bolivia.

The great distance from this country of the Andean resorts requires that every case which it is proposed to send there should be made the subject of special consideration.

The favourable changes observed to occur from a residence at a high altitude are that the cough and expectoration either diminish or disappear, the strength and appetite increase, and there is generally some gain of weight. At the same time examination of the chest reveals changes in the physical signs, which indicate that the disease has either become quiescent or undergone complete arrest. The most satisfactory change is the disappearance of the adventitious sounds.

Dr. Theodore Williams has shown that an enlargement of the thorax follows a residence at a high altitude, and that the increased resonance of the percussion note observed is not limited to the site of the contracting lesions, where no doubt it is in part due to the production of emphysematous changes in the surrounding lung, but that it occurs in all regions of the thorax.

If the contraction of a cavity is followed by thickening of the overlying pleura, the percussion note does not improve in resonance, but becomes [illegible] dull.

Dry warm climates.—*Egypt.*—The climate of Egypt, especially where the influence of the desert is chiefly felt, is characterised by purity and dryness of the atmosphere, warmth, abundant sunshine, very little rain, and a great difference between the night and day temperature. The disadvantages of the climate are the occurrence at certain periods of hot dust-laden winds from the desert, and at others of cold winds and the prevalence of intestinal disorders.

The reputation of Egypt as a resort for pulmonary invalids was gained chiefly by the good effects observed to follow a voyage up the Nile by *dahabeyah*, and that is probably still the best way in which an invalid can spend his time. The steamers, which now make the journey in a short time, present advantages to the tourist only. The chief health resorts are Mena House, a short distance from Cairo, Helouan, Assouan and Luxor. We have known advanced cases of the disease derive great benefit at Helouan, but patients must be prepared for a period of absolute dulness and must be content to spend the greater part of the day in the desert. Pulmonary invalids should absolutely avoid Cairo.

No case should be sent to Egypt unless there are clear indications that it is unsuitable for treatment at a high altitude, and, owing to the distance and the expense of the journey, and the high cost of living, only patients with ample means are able to enjoy the luxury of wintering there. The invalid should, as a rule, arrive in Egypt about the middle of November and leave about the middle of April.

The cases of tuberculosis most likely to derive benefit from a residence during the winter months in such a climate are those complicated by bronchitis and emphysema, but with a limited area of infiltration. The presence of albuminuria in an otherwise suitable case is rather an indication than otherwise for this climate.

The Riviera.—The climate of the French and Italian Riviera is suitable for some cases in which the disease is more advanced, or when, from one or other of the various causes already stated, the high altitudes are contra-indicated.

During the winter months the mean average temperature is higher by from 8° to 10° F. than that of this country, whilst the amount of sunshine and the number of days without rain is far greater.

The disadvantages of the Riviera climate are the great difference between the sun and the shade temperature, the marked fall of temperature immediately after sunset, and the prevalence, especially at certain times of the year, of winds which prove extremely trying to the invalid—conditions which necessitate the greatest care in order to avoid exposure to chill.

The most sheltered place is the eastern bay of Mentone, but the almost complete protection from wind which is there obtained, combined with full exposure to the sun, produces a climate which is only suited to patients of rather feeble vitality, and which exercises an enervating effect upon the more robust.

CHAPTER XXXVI

SURGICAL TREATMENT OF PULMONARY CAVITIES

INCLUDING THAT OF FOREIGN BODIES IN THE BRONCHI

PAGE
- Classification 417
- Acute abscess following pneumonia 417
  - Steps of operation 418
    - Anæsthetic 418
  - After-treatment 420
  - Prognosis after operation 421
  - Cases unsuited for surgical treatment 421
- Bronchiectatic cavities 421
  - Operation 422
- Tubercular cavities 423
- Chronic non-tubercular cavities 425
- Foreign bodies in the bronchi 425
- Treatment 426
- Conclusions 427

Historical.—In a work like the present, which is intended to be practical, it is probably unnecessary to enter into the history of the subject of surgical interference for the relief of cavities of the lung. I have discussed the subject fully in some lectures delivered at the Hospital for Consumption and Diseases of the Chest at Brompton, which are reported in the first volume of the 'Lancet,' 1887, and those who desire still further to investigate the matter will find a very extensive bibliography in Riedinger, 'Verletzungen und chir. Krankheiten des Thorax' (Deutsche Chirurgie Lief. 42), 1888. Here it may be briefly stated that Sir Edward Barry advocated the opening of phthisical cavities in 1726, and it is said that before his time Baglivi recommended the incision of pulmonary cavities in 1714.

Dr. John Hastings and Mr. Starks treated some cases in 1830. A few years later (1855) Dr. Horace Green recommended the injection of bronchiectatic and tubercular cavities by means of a tube passed through a bronchus.

In 1873 Dr. F. Mosler of Greifswald described a process of injecting by means of a cannula and trocar passed through the when the disease has attacked the glands and bones than when the lungs are chiefly involved.

There is, at the present time, a general agreement that no patient with advanced lesions should be sent for a long sea voyage, and also that in the early period of the disease the majority of cases derive far greater benefit from a residence in one of the 'open-air' sanatoria or at a high altitude.

Vigorous patients with limited or quiescent disease, who have a special liking for the sea, may make the voyage round the Cape to New Zealand or Australia, or they may proceed by the Suez Canal to India, and thence to China and Japan.

Notwithstanding statements to the contrary, we believe it is, as a rule, better for the patient to take a passage in a well-appointed steamer, in which every provision is made for his comfort, than in a sailing vessel.

Mineral waters.—The warm sulphur springs of Eaux Bonnes and Cauterets are very useful in catarrhal conditions, and in some cases of quiescent tubercular disease of the lungs presenting such complications, they may be resorted to with advantage. The sulphur springs have also a reputation for the relief of glandular tuberculosis. The arsenical waters of La Bourboule, and Mont Dore, and some of the common salt waters, particularly those of Ems, are of service in cases of fibroid or arrested tuberculosis associated with emphysema and bronchial catarrh.

J. K. F.

time the surgeon should proceed to make an external opening. It must not be supposed, however, that this is a perfectly simple operation; the localisation of the cavity is often extremely difficult, principally because the physical signs of consolidation are apt to mask those which are caused by the cavity, but also because the abscess is often so deeply placed, that it practically gives rise to no physical signs of a definite character. It has therefore not unfrequently happened that, even after the discovery of an abscess and the introduction of a drainage tube, the post-mortem has revealed other cavities in the same or even in the opposite lung.

Steps of the operation.—After endeavouring by careful physical examination to *determine the whereabouts of the abscess*, the first point is to introduce the needle of an aspirator, and if the matter be not discovered at once, it should be introduced again and again, at any place at which the physical signs may render the exploration justifiable, even although they do not seem to indicate the presence of a cavity. Sometimes, for example, it may be found that there are marked signs of a cavity at the lower part of the lung whilst the abscess is situated near the apex of the lower lobe. It must not be forgotten that in puncturing a lung which is charged with highly septic material a risk is run of defiling a pleura in which no adhesions have taken place, but this danger is not so great as it is in the case of bronchiectasis.

Supposing the exploration to have resulted in the discovery of pus the patient is anæsthetised and the operation is proceeded with. The *administration of an anæsthetic* to patients who are expectorating fœtid material is fraught with the danger of encouraging the inspiration of this material into other parts of the lung, in addition to that of the chance of suffocation, which is a real danger, whatever the nature of the fluid may be. This is the reason for making the exploratory puncture before giving the anæsthetic, but obviously the advisability of doing so must depend on the particular case, it being impossible in dealing with nervous patients, especially if extremely ill, to carry out such a series of punctures as may be required before the surgeon feels justified in giving up the search as hopeless.

As in the case of operating on empyemata, the best anæsthetic I think is chloroform, and the anæsthesia should not be carried to a very advanced degree. The objection to ether is that it occasions sometimes a copious bronchial secretion, which in itself interferes with respiration, and which, when added to the fluid expectorated, may become extremely dangerous to the patient. The anæsthesia should not be very complete, because it is desirable that the patient should be able to cough up the accumulated fluid. Of course this cough is a great inconvenience to the surgeon, but it is an inconvenience that he must put up with and learn to become accustomed to. From the same point of view it is advisable that the patient should not be turned too far on to the healthy side. In order to render the operation possible, it may sometimes be necessary to draw the patient beyond the edge of the table, whilst the surgeon sits upon a low stool (fig. 140, p. 601), and in very bad cases it may be wisest to place him altogether on the diseased side, and standing upon the opposite side of the patient, to operate, as it were, from behind (fig. 141, p. 602.) These last remarks refer to the treatment of each of the three kinds of pulmonary abscesses.

Having ascertained the presence of pus, it is not wise to withdraw the needle, but it should be kept *in situ* as a guide. This is easily said, but not so easily done. The assistant to whose care the needle is given over is liable to have his attention distracted, and if he looks aside for a moment he will very likely, by slightly moving his hand, either withdraw the needle, or insert it too deeply, or bend it to one side or the other; and the same result may easily follow a chance touch from the hand of the operator, or from that of the assistant who is spongeing. An incision is then made as for the resecting of a rib, but seeing how often it is found that the excision of more than one rib is required, and also that after the opening is made it is the very lowest part of the cavity that has been opened, it will be usually wisest to make a vertical incision, rather than the oblique one which was recommended for cases of empyema. A piece of rib, probably above the intercostal space which has been perforated, is then removed, in doing which great care should be taken not to injure the pleura, because, although the presence of adhesions is very likely, it is not certain that they will be found. The cleverest physician cannot say whether adhesions will be met with or not, nor can he be quite sure how far the solidification of the lung will prevent that organ from falling away from the chest walls in case the pleura should be healthy. It therefore behoves the surgeon to proceed as if the lung was not adherent; this has the incidental advantage that if it should actually be the case he will feel satisfied that he has not admitted septic material from the abscess into the healthy pleura. Having removed portions of one or two ribs, as the case may be—and he will most frequently find that one is not sufficient—a series of stitches should be placed with a full curved needle fixed to a Hagedorn's or any other suitable form of needle-holder. The stitches must pass through the pleura into the affected lung. A single row is quite sufficient, but a double row is of course safer. From six to eight stitches, including a circle of from one and a half to two inches diameter, are usually enough, and can be introduced in a very short time. Stitches may be made of silk or fishing gut or catgut, and they should enter about one half to three-quarters of an inch into the lung substance. The pleura is now to be incised, and it will be found, even if there be no adhesions, that if the stitches have been properly introduced a pneumothorax will not result; should, however, air suck in at one part of the incision, further stitches must be used until the whole circumference has become air-tight.

As to the *method of incising the lung* various opinions have been held and many devices have been invented. The risk in connection with it depends upon the possibility of injuring one of the large pulmonary vessels, our knowledge of the anatomy of which cannot be sufficiently accurate to enable us to avoid them with certainty. Some reference to their description on pp. 42 and 46 will give a general idea of where the main trunks are to be found, but the branches of the artery and the tributary veins are so large in all parts of the lung that this knowledge is not of much practical importance. In every part of the lung, indeed, vessels are to be found large enough to bleed to death from.

Fortunately, however, the hæmorrhage can usually be stopped by the pressure of a plug. It is from fear of hæmorrhage that many have advocated the use of the actual cautery; it is not a procedure with which I am enamoured. The charring of the tissues is confusing to the finger which is introduced in order to explore the cavity, and, further, the destruction of the tissues encourages a subsequent foul condition of the wound. If the abscess be obviously near the surface, there can be no doubt that the best way is to open it by means of a pair of sharp-pointed forceps (sinus or dressing forceps) introduced alongside the guiding cannula. After they have reached the cavity the blades are opened, and, the cannula being withdrawn, the finger is gently insinuated along the same track, which its passage enlarges sufficiently to permit the introduction of a drainage-tube of a suitable size. I have been in the habit of adopting the same plan for opening abscesses at a greater depth, and have not yet met with any insuperable difficulty in so doing, though I have sometimes been obliged to plug the wound. The finger explores the cavity as far as possible, and, if it is found easy of access, some iodoform or boracic acid or other suitable antiseptic powder may be introduced. The wound of the soft parts should be most thoroughly treated with chloride of zinc (40 grs. to the ounce), and plugged round the tube with some lint which has been soaked in the same solution and dusted with iodoform. The dressing is then applied, and, if the matter be very foul and frequent changing is likely to be required, some simple application, such as fomentations of boracic lint covered with wood wool or oakum, will be found most convenient for the purpose.

After-treatment.—In the after-treatment of the case no effort should be spared to sweeten the cavity, but injections of fluid are inadmissible, as such would be sure to enter the trachea and cause troublesome coughing; their place must be taken by powders, such as those referred to above. The tube must be frequently washed, but it is better to leave it for a day or two *in situ*, so that the track by which it has entered the cavity may not be lost. The tube, it may be said, must reach to the end of the cavity, and may be provided with holes in the part that corresponds to the cavity. There is much more chance of the tube giving trouble than in the treatment of empyema, because, just as happens so frequently in cases of hepatic abscess, the process of cure is associated with more or less contraction of the organ, which changes the direction of the sinus and the position of the cavity. It thus is often advisable to prevent the slipping of the tube by placing a piece of trustworthy plaster across the deeper part of the dressing. The tube must be retained until the external discharge is reduced to a very small quantity after the complete stoppage of expectoration.

Prognosis after operation.—It is obvious, from what has been said about the pathology of gangrene of the lung in pneumonia, that no very large percentage of recoveries can be expected to follow surgical interference, seeing that death may follow from extension of the pneumonia, from blood-poisoning, or from hæmorrhage. While, therefore, too much must not be expected from the evacuation of the abscess, still, the chance of recovery being so slight without interference, I have no hesitation in recommending a course of procedure such as has been described. The least hopeful cases are those in which the abscess is most acute, in which there is reason to suspect affection of the opposite lung, and in which the expectoration has the dark brown colour with that particularly filthy smell which is so characteristic of the disease. The most promising cases are those of a less fulminating character, in which only one lung or, better still, only one lobe of one lung is affected, in which the physical signs are localised, and in which the expectoration, if any, is of a distinctly purulent character.

Cases unsuitable for surgical treatment.—There is a class of case which must be considered here that is altogether beyond the reach of surgery. It comprises those in which collapse and gangrene follow upon the accumulation of septic material which has collected in the bronchi, either where one bronchus is pressed upon by a tumour, aneurysmal or otherwise, or where it has entered the trachea as the result, say, of cut-throat or tracheotomy. If this state of things has been clearly made out, no good can come of surgical interference. The mere exploration of such cases with the needle has been followed by the development of acute empyema. I once had to deal with such a case in which the history had not been explained to me at the time when I had to operate; the pus of the empyema was highly offensive, but I did not discover till some time after at the post-mortem examination that a large aneurysm of the aorta was projecting into the pleura, which might conceivably have been injured at the time of the operation. I need hardly say that since this experience I always endeavour to obtain from the physician a strong opinion as to the possibility of the presence of this extremely dangerous complication to a surgical operation in cases where there seems to be the slightest likelihood of its presence.

2. **Surgical treatment of bronchiectatic cavities.** The next class of cases to be described is that of *bronchiectasis*, the pathology of which has been discussed on page 125 *seq*. It remains only to point out, from a surgical point of view, a fact which will be returned to later on—namely, that bronchiectasis is not unfrequently the result of a foreign body having been inspired, and also to indicate the mechanical difficulties that are presented to effectual drainage from the very nature of the complaint. From whatever cause the bronchiectasis may have arisen, the cavity is always of a

complicated nature, and, although communications may form between contiguous parts of a dilated bronchial tree, it must not be forgotten that as a rule the opening of each dilated bronchus into the main channel takes place at the upper part of the cavity. It follows from this that an incision into one part of such a branched cavity can do nothing in the way of draining the others, and the advisability of resorting to any surgical interference depends very much upon whether or not the main part of the symptoms are caused by one cavity of considerable size. Soon after I became surgeon to the Brompton Hospital, I operated on a certain number of cases of bronchiectasis at the request of my colleagues. In not a few of them it was possible to open a considerable cavity, but in the greater number there were not only physical signs distributed over a very extensive area, but they were occasionally heard to a less extent upon the other side of the chest. The result, I am sorry to say, was not encouraging. It frequently happened that the symptoms were decidedly improved for a time, or rather it should be said that the most troublesome symptom—that of copious expectoration—was to some extent alleviated; but in almost every case, as the disease advanced, fresh cavities took the place of the one that had been drained, the amount of expectoration increased, and various maladies of an intercurrent nature, not directly attributable perhaps to the operation, made their appearance. The patients frequently suffered from hæmoptysis, more than one developed cerebral abscess, and some died of acute septic nephritis. I cannot therefore encourage surgical interference in cases of bronchiectasis pure and simple, unless it can be shown that there is a high probability of the disease being confined to a very small part of the lung. In a few such cases recovery may take place. Dr. Williams and myself reported one such in the 'Transactions of the Royal Medical and Chirurgical Society' for 1886 (vol. lxix.), and I take this opportunity of saying that some years afterwards the patient died of heart disease, and I had the opportunity of verifying the correctness of Dr. Williams's diagnosis, and seeing that it was not, as some might have thought from the account of the case, an empyema.

Operation.—Should, however, the operation be attempted, the steps of it do not differ from those described on page 418. It must be remembered, notwithstanding all assertions that may be made to the contrary, and deductions from post-mortem examinations, that the surgeon seldom meets with adhesions in these cases; and not only is this so, but if the pleura has been opened before stitching up the lung, as was done in some of my early cases, the lung recedes far away from the opening, and feels to the fingers perfectly healthy, so that it is impossible to make out by the touch the position of the abscess. It is therefore essential to fix the lung before opening it, and if by chance this precaution be omitted and air be allowed to enter the pleura, there is nothing for it but to close the wound and wait for the absorption of the pneumothorax before making another attempt. The feeling imparted to the finger by the interior of a bronchiectatic cavity is quite different from that of an abscess resulting from the breaking down of the lung. In the latter case an irregular spongy mass is felt which is easily broken down; in the former the lining is perfectly smooth and firm, and the cylindrical shape of the cavity is, moreover, usually easily determined. It is not improbable that manipulation with the finger or the contact with the drainage tube will set up a fit of coughing. The after treatment of these cases does not call for any special comment. The discharge is usually copious, sometimes, but not always, offensive, and consists of sticky muco-pus. Should it be found that the patient has not derived much benefit from the operation, it may be thought wise to remove the tube, when the external opening will probably close, and the patient will be left no worse off than before.

When an external incision has been made into a cavity communicating with a bronchus by a large opening, a curious phenomenon is generally observed. Not only is the sound of the air entering and leaving the chest of a loud whiffing character, different from that which is heard in the case of cavities similarly opened that do not communicate with bronchi, but the communication is sometimes so free that the patient can breathe through it whilst keeping the mouth and the nose closed. I have seen cases where this could be done for quite a considerable time, and it could be determined by the stethoscope that air was entering freely into the opposite lung. Under such circumstances, when the dressing is removed, the patient is unable to talk above a whisper. Sometimes, indeed, he cannot do so even when the dressing is applied. Should this be the case, a small plug of wool placed in the orifice of the tube will be found a great comfort to him.

3. **Surgical treatment of tubercular cavities.**—The surgical treatment of *tubercular cavities* does not appear up to the present to have called forth much enthusiasm, and yet it might have been thought (though here I am only expressing my own private opinion) that the same treatment would have been applicable to a tubercular deposit in the lung, as proves successful in almost every other part of the body, however inaccessible its position or however vital its functions. This excites the greater surprise when one thinks of the amount of inconvenience caused to the patient by the constant expectoration, and of the fact, so often insisted upon before, that profuse expectoration is frequently the means of disseminating mischief through previously healthy portions of the lung. It would therefore be rash to prophesy that there is not a future for this branch of therapeutics. Probably several reasons combine to dissuade physicians from calling in surgical aid. In the first place, it very commonly happens that, by the time a cavity of sufficient size to be diagnosed by physical signs has formed, the disease has already advanced so far as to be practically beyond the hope of relief, and almost certainly has reached such a stage as

to render the possibility of removing the whole of the tubercular deposit impossible. Secondly, it is just those cases of single, stationary, or very slowly advancing cavities which promise the best field for surgery, that not unfrequently dry up and cicatrise. Thirdly, there is the fact that the expectoration from tubercular vomicæ, though, no doubt, often very troublesome, does not form so distressing a symptom as it does in some other cases of pulmonary abscess.

It must not be supposed that no plan of surgical treatment has been suggested or carried out. Whole series of cases have been described by Dr. Shingleton Smith and others, in which antiseptic fluids (a favourite one being a solution of iodoform in ether) have been injected by means of a syringe introduced through an intercostal space. Of this plan of treatment I cannot speak from personal experience, and can only say that the use of these *intrapulmonary* and also of *intralaryngeal injections* of various fluids is reported to have been attended by a varying degree of success.

The most heroic method of treatment yet suggested comes from Italy,[1] and it has been followed up since then in Germany, the treatment being to remove the affected portion of the lung. Biondi made a number of experiments in rabbits, cats, and dogs, removing from them the whole of one lung, and he showed that, although a large proportion of the animals died, a certain number survived, and some in whom tubercle had actually developed in the lung as the result of inoculation, not only lived but remained afterwards free from the disease. These observations are of great importance not only in connection with the study of pulmonary tuberculosis, but also in connection with the question of the removal of tumours of the lungs (see page 489). As to this line of treatment also I have no personal experience.

The most rational treatment of tubercular cavities appears to me to be the opening and draining of them in the same way as other cavities of the lung are treated. The first cases that were reported, about 1845, did not give very satisfactory results, and at the Congress at Wiesbaden in 1883, the consensus of opinion was strongly against the opening of tubercular cavities, until, as it was said, a trustworthy specific tubercular bacillicide should be found. Writing in 1887, I said that though, on the whole, agreeing with that opinion, I could not help thinking that in a certain number of cases with single apex cavities and with troublesome cough, not only might the symptoms be relieved, but the danger of transferring the mischief into the other lung might be to some extent lessened. I can see no reason for altering that opinion, but I have never yet been asked to operate upon a large tubercular apex cavity. I have, however, treated several cases of tubercular cavity in other parts of the lung; one of them at least healed, though the patient died subsequently of pulmonary tuberculosis; but in others the sinuses remained open, and the patients have appeared to be benefited by the escape of a certain amount of the discharge externally.

---

[1] 'Estirpazione del Polmone,' per il Dr. Biondi, *Gior. Internaz. d. Sc. Med.*, N.S. iv. 759.

Not long ago my colleague Dr. Acland requested me to explore the chest of a man who was bringing up a large quantity, 20 to 30 ounces, of extremely fetid expectoration, which contained tubercle bacilli, and in whom physical signs suggested strongly a large cavity. This, however, the exploration failed to detect, and at the post-mortem examination it was found that the affected part of the lung was riddled with small gangrenous cavities. I have now in hospital a young woman who is the subject of syphilis; she developed physical signs at the left base with pyrexia, and other symptoms which pointed either to an empyema or to a pulmonary cavity. Before long the abscess, which was in the lung, burst into a bronchus, but did not discharge sufficiently freely. I opened it by an incision in the side and inserted a long tube. She now keeps comparatively well as long as the sinus remains open, but if the tube is taken out the temperature rises. The pus is copious and muco-purulent, and contains tubercle bacilli.

4. **Chronic non-tubercular cavities.**—About this class of cavity nothing need be said except that they are occasionally met with, and that the treatment of them calls for no special notice. They are, however, the most promising of all for surgical interference. Some of them are caused by the rupture into the lung of abscesses which have originated elsewhere, for example in the liver.

5. **Cavities caused by the presence of a foreign body in a bronchus.**—It is practically useful to consider separately the results that are caused by very smooth and unirritating foreign bodies, and those that are caused by rough and irritating ones—for example, by such things as beads, or peas, or tracheotomy tubes and such objects as bits of straw, or nut-shells, or bones. The immediate effect of the lodgment of a smooth foreign body may be very slight. I have known the shield to come off a new vulcanite tracheotomy tube soon after its introduction; and notwithstanding very careful physical examination, it was impossible for some days to be quite certain that the tube was in the chest, and that it had not been lost in the patient's bed. A piece of grass or a sharp bone, on the other hand, will give rise to immediate attacks of spasmodic cough, a result which has sometimes led to the error of supposing the disease to be whooping-cough, and the story of the inhalation of the foreign body to be apocryphal. I would most strongly insist upon the inadvisability of rejecting a patient's account of 'swallowing' a foreign body. It is an accident that forcibly impresses itself upon his mind. If the immediate effect has been slight, he may indeed have forgotten it; but if he remembers the occurrence at all, he is not very likely to be mistaken about it. Within a short time of the inspiration of the foreign body, whether it be rough or smooth, pneumonia will probably result; but the later effects vary very much in the two

cases. If a piece of grass or an ear of wheat have passed into the bronchus, it may possibly perforate the lung and travel to a distant part and ultimately make its way through the skin; but if this does not take place, it will set up acute changes consisting of dilatation of the bronchi, of breaking down of the lung, and most probably of burrowing abscesses. If the body be a smooth one, the changes are much more chronic, and consist of dilatation of the bronchi with a certain amount of consolidation of the lung. Patients may go on for years with this condition of things, and if they have forgotten or do not mention the cause of their complaint, it is very likely that it will be taken for bronchiectasis arising from one of the more common causes. It is for this reason that I would impress upon the reader the importance of inquiring in every case of unilateral bronchiectasis as to the possibility of its having arisen from the inspiration of a foreign body.

It is most important to remember that a very short residence of a foreign body in a bronchus may give rise to an amount of bronchiectasis from which recovery is impossible. I have notes of the case of a boy who, when seven years of age, had a small ivory knob in his left bronchus for eight weeks in the year 1891. He has now diffuse bronchiectasis of the lower part of the left lung, which appears to be beyond surgical treatment, and shows no sign of any improvement. In another case the vertebra of a rabbit was impacted in a bronchus in December 1887. He developed well-marked bronchiectasis, upon which I operated in the hope of finding a foreign body; but the bone was not coughed up till some time after the operation—namely, in April 1889. He is now, in 1896, very much improved, but there remains a cavity at the base of his lung which has never quite stopped secreting, and is liable to attacks of acute inflammation. There was, at University College Hospital, under my care, a boy who had the peg of a pegtop in his lung for years. After it had been expelled through an opening which I had made, but through which I had failed to reach it, he was left with a cavity which continues to discharge a considerable amount of pus, and he still has severe attacks of hæmoptysis.

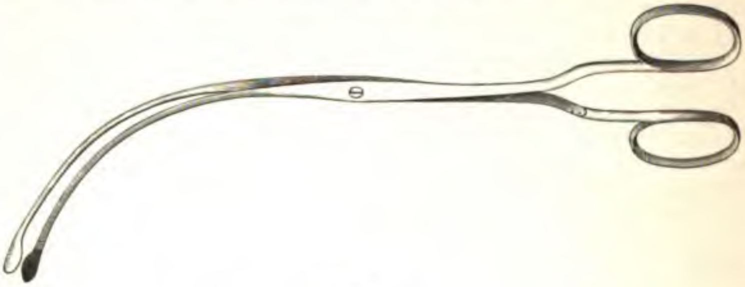


FIG. 111

Treatment.—Seeing, therefore, the extreme improbability of a favourable result in chronic cases, I would urge most strongly that every case in which there is good reason to suppose the presence of a foreign body in the bronchus should be dealt with without delay. It is, I think, quite unjustifiable to wait for symptoms to declare themselves, as these symptoms may indicate the presence of mischief from which recovery is impossible. The right course of procedure is to perform tracheotomy, and to endeavour to extract the foreign body by means of forceps (the pair shown in the figure (fig. 111), which are so arranged that when the points are slightly separated the combined width of the blades slightly diminishes, are very useful for the purpose), or by means of probangs, or by coin-catchers, or—that which will be found very useful—a piece of stout silver wire bent as indicated in fig. 112, which can easily be directed to the bronchus of either side. If this be unsuccessful, the patient may be inverted and shaken—a procedure which should not be attempted before tracheotomy has been performed. Subsequently, if acute suppuration have occurred, there can be no doubt of the right course to pursue; but the surgeon will be unusually fortunate if he meets with the foreign body, though a careful search for it should not be omitted. These cases are not unlikely to prove rapidly fatal.

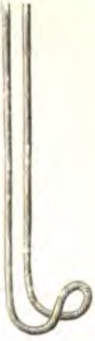


FIG. 112

In the chronic cases it is right to endeavour to determine the position of a large cavity, and to open this as described on page 418. It should then be explored as far as is possible with probes and forceps, and if these fail tracheotomy should be performed, and the same course pursued as in cases of recent impaction. Another method, which was employed in the case of the boy who had swallowed the peg of the pegtop, is to make an incision into the pleura behind the place at which the abscess had been opened, and to feel over the back and under surface of the lung. It was not successful, but I can imagine that another time it might prove to be of value. This case also proved the advisability of retaining the tube for a long time, for it will be remembered that it was through my opening, and not through the trachea, that this large foreign body was at last expelled.

Conclusions.—It may, I think, be safely stated: I.—that gangrenous cavities should always be sought and if possible opened; if the operation be successful and the opening free, the prognosis is not bad. II.—The same treatment should be applied to abscesses caused by the rupture of purulent collections from other parts into the lung, at least as regards the pulmonary complication. III.—Abscesses caused by the presence of foreign bodies should be opened, and if the body be not found, it must be remembered that, if of any considerable size, it probably lies not more than three inches from the middle line. If possible, these cases should be treated early by tracheotomy and inversion. IV.—Bronchiectatic cavities when single—a very rare condition—may be cured by operation; when multiple—a v common condition—they offer a very small chance of relief by our present methods; still it may be wise in some cases to make an attempt to open the main cavity, if such is to be found, but only after it has been ascertained that the lung is adherent to the chest wall. V.—Basic tubercular cavities sometimes require the same treatment as is applied to other pulmonary cavities. VI.—It is not usual to open apex cavities, though this may be justified by an extremely harassing cough. Such operations cannot be expected to be curative. VII.—Tubercular cavities may be treated by injections, either through the larynx or through the chest wall, but intralaryngeal injections appear to be practically valueless, and little is to be hoped from intrapulmonary injections.

R. J. G.

CHAPTER XXXVII

PULMONARY SYPHILIS

	PAGE
Hereditary syphilis	430
Morbid anatomy	430
Association of congenital syphilis and pulmonary tuberculosis	432
Acquired syphilis	434
Pathology and morbid anatomy	434
Illustrative cases	436
Symptoms	449
Physical signs and diagnosis	450
Prognosis	451
Treatment	452

Our knowledge of the anatomical characters and clinical history of syphilitic disease of the lungs is still very incomplete notwithstanding that much has been written on the subject. This is due in part to the rarity of the affection, but chiefly to the difficulty until lately experienced in distinguishing between the lesions of syphilis and those of tuberculosis.

Up to the date of the discovery of the tubercle bacillus it was very often impossible to determine with certainty during life whether a given case of pulmonary disease was tubercular or not; and, after death, appearances which some considered to be distinctive of tubercle were by others said not to possess that significance. Now, however, that we possess a test for tubercular lesions it may reasonably be hoped that the whole subject of syphilitic disease of the lungs will be placed upon a secure foundation.

That the disease is of rare occurrence is a fair inference from the fact that the museums of the London hospitals and of the Royal College of Surgeons, all of which the writer has recently visited, contain only twelve specimens which are believed to illustrate syphilitic lesions of the lungs; and of these two may be excluded, as either not of that nature, or of a nature so doubtful, that in the present state of our knowledge they are inadmissible as evidence. None of these specimens is from a case of congenital syphilis.

Morbid anatomy.—The following pulmonary lesions have been attributed to syphilis: (*a*) gumma; (*b*) white hepatisation (Virchow, Weber), or 'epithelioma of the lung' (Lorain, Robin) (*c*) grey infiltration (Welch, Pancritius); (*d*) lobular pneumonia or bronchopneumonia (Förster, Welch); (*e*) fibroid induration; (*f*) changes in the lymphatics (Hermann Weber); (*g*) a destructive disease, the so-called 'Syphilitic Phthisis.' It will be convenient to consider separately the lesions of the hereditary and the acquired disease.

Hereditary syphilis.—The pulmonary changes in hereditary syphilis may be either circumscribed or diffuse; to the former the term 'gumma' is applied; the latter are classified under the head of 'pneumonia.' It is, however, far more common to find the two changes associated than to meet with either separately.

A. *Gumma.*—As this lesion is of comparatively rare occurrence in congenital syphilis, and when present does not differ either in appearance or in microscopical structure from that found in the acquired disease, a more distinct picture of the morbid anatomy of the two affections will be obtained by describing it under the latter heading.

B. *Pneumonia.*—Two separate lesions are included under this heading, namely, 'white pneumonia' and 'interstitial pneumonia;' but of these it must again be stated that they occur more often in combination than apart.

(*a*) *White pneumonia* (Virchow, Weber), *Epithelioma of the lung* (Lorain, Robin).—This lesion, which in its true form is rare, is found only in the lungs of still-born children, or of such as have survived their birth a very short time. Other unmistakable signs of congenital syphilis are usually present, and gestation has in such cases seldom proceeded to the full term.

It is a diffuse change affecting a lobe either as a whole or in part; or one or both lungs may be completely consolidated.

In still-born children the affected part is bloodless and airless; and even if force be used it may be impossible to inflate it; but in infants several days old the lung always contains some air.

The lung is much increased in size and its surface may be marked by the ribs. It is solid, dry, white, yellowish, or greyish-white in colour; but sometimes presents a reddish marbled appearance. The section differs from that of an ordinary pneumonic lung in that the granular appearance characteristic of the latter is absent, the surface being smooth and somewhat shining.

On microscopical examination in true cases the interstitial tissue is not increased. The alveolar walls are thickened, and the small bronchi and the alveoli are filled with masses of cells of which some are round and others have more or less the character of epithelial cells: the cells are for the most part undergoing fatty degeneration and are beginning to break down. The alveoli are markedly enlarged. The colour of the affected area is due partly to the above changes, but in part also to diminished blood-supply the result of pressure upon the capillaries. The lung tissue surrounding the consolidated part may show some degree of emphysema. Ecchymoses may be present in the pleura, pericardium, and thymus gland; but these appearances are probably incidental to the mode of death.

The bronchial glands are as a rule enlarged and on section dense, from a new formation of fibrous tissue enclosing cells arranged in a concentric manner.

White pneumonia is a lesion of purely pathological interest, as owing to the filling of the alveoli with cells, the subjects of it, if not still-born, are unable to maintain the respiratory function for any length of time and soon succumb.

(*b*) *Interstitial pneumonia.*—This is the most common pulmonary manifestation of hereditary syphilis; but it occurs more frequently in association with some of the changes described under 'white pneumonia' than as a purely interstitial lesion. In its true form it is distinguished by a small-celled infiltration of the interalveolar connective tissue, the alveolar epithelium remaining unaffected. This change may be present to a very varied extent. In some cases lungs so affected appear normal to the naked eye, the lesion being only discoverable on microscopic examination.

In well-marked cases the lungs are large and hard and of a pale or dark greyish red tint. The change may be present throughout the organs, or a single lobe or portion of a lobe may be alone affected. To the naked eye the lung tissue presents a decidedly coarse appearance. On microscopical examination a marked increase is seen in the interalveolar and interlobular connective tissue, which forms broad meshes, including small spaces wherein the alveoli are either crowded together or completely obliterated.

In some cases the interalveolar meshes appear to consist of a dense capillary network, the vessels being dilated and tortuous. Around the vessels and bronchi there is a marked increase of the connective tissue, and the tunica intima of the small arteries is thickened. The alveolar epithelium may show desquamative changes, and brown and yellow pigment granules may be present.

Interstitial pneumonia is often found in association with congenital syphilitic lesions in the skin, with interstitial hepatitis and changes in the epiphyses; but it is also found in cases in which gummata are present in the lungs, liver, and other organs. The change begins during fœtal life, and at birth may have affected the lungs extensively. In such cases life is of short duration and death occurs from asphyxia, as is shown post-mortem by the frequent presence of ecchymoses in the pleura, pericardium, and thymus gland. When the change is less advanced at birth such children may die at a later period by a slow process of carbonic acid poisoning, the first sign of which may be that a child previously fretful and noisy becomes quiet.

In cases in which the other organs are healthy, or nearly so, life may be prolonged for months or years; such subjects are, however, specially liable to acute disease of the respiratory organs, such as pleurisy, acute bronchitis, and broncho-pneumonia.

From the above description it will be seen that the morbid processes concerned in the production of the gummatous and diffuse changes found in the lungs of syphilitic children chiefly affect the connective tissue and small arteries. They are (i) a round-celled infiltration and proliferation of the interlobular and interalveolar connective tissue, originating in the cellular tissue around the bronchi, and leading to marked thickening of the framework of the lung. (ii) An isolated perivascular cell proliferation, which begins around the small arteries, and is accompanied by changes in the tunica intima (Hochsinger). Both the periarterial and peribronchial granulations may occur as separate nodules or node-like foci; or they may be diffused over large portions of the lungs. A well-marked desquamation of the alveolar and bronchial epithelium is almost always present, but it is quite a secondary process.

In the account here given of the pulmonary changes found in hereditary syphilis the descriptions of Heller,[1] Spaundis,[2] and Hochsinger have been followed, and to those authors the writer desires to acknowledge his indebtedness.

Association of congenital syphilis and pulmonary tuberculosis.—Syphilis, by lowering the resisting power of the individual, may predispose to tuberculosis; and it has recently been shown by Hochsinger[3] that the virus of syphilis and tuberculosis may be jointly transmitted from parent to offspring.

This observation is of much importance, and throws a new light upon the nature of the pulmonary lesions found in infants the subjects of congenital syphilis. It has often hitherto been assumed, on evidence which is now proved to be insufficient, that such lesions are of syphilitic origin, whereas it is clear that they may be due to an associated tubercular infection.

In three infants suffering from congenital syphilis, and presenting symptoms of infiltration of the lungs, the pulmonary disease was found post-mortem to be due to tuberculosis and not to syphilis. Tubercle bacilli were found in the lungs in all the cases.

The first case was observed in 1891 in a child not quite three weeks old; the second in 1891 in a child twenty-four days old; the third in 1893 in a child eleven weeks old.

Case I.—Anna B., æt. nearly three weeks. The parents had been married nine years. The father acquired syphilis shortly before marriage. The mother died from pulmonary tuberculosis three months after the birth of the child. The first and second children of the marriage were stillborn; the third and fourth died during the first week; the fifth and sixth were living, ages four years and two years respectively. The case of the seventh child is here described. From the time of birth she was sickly and suffered from nasal obstruction, snuffles, and dyspnœa. Râles were present in the chest. At the end of the second week a bullous eruption appeared on the nates. The child presented all the ordinary external signs of congenital syphilis, and was shown at the Vienna Dermatological Society as a case of gummatous disease of the viscera.

---

[1] Heller, 'Die Lungenerkrankungen bei angeborener Syphilis,' *Deutsch. Archiv f. klin. Med.* Bd. xlii. S. 159, 1888.

[2] Spaundis, 'Ueber congenitale Lungensyphilis,' *Inaugural Dissertation*, Freiburg, 1891.

[3] Hochsinger, *Wiener Med. Blätt.* Nos. 20, 21, 1894.

On examination of the chest there was marked dulness on the left side from the angle of the scapula downwards, with bronchial breathing over the dull area. The respiratory murmur was harsh over both lungs, with rhonchi and coarse râles. The spleen was enormously enlarged, extending as low as the anterior superior spine of the ilium; the liver could be felt four fingers' breadth below the costal margin, it was hard and the edge was rounded. The diagnosis was pulmonary and visceral syphilis. Mercurial treatment was prescribed. The child died on the thirty-first day after birth. On post-mortem examination the internal organs were found extensively infiltrated with tubercle. Both lungs showed tubercles varying in size from a miliary granulation to a walnut. A nodule as large as a hen's egg occupied the right middle lobe. The left lobe of the liver was almost completely replaced by a caseous nodule; numerous tubercles studded the right lobe. The spleen was enlarged to nearly four times its normal size and contained similar deposits. Tubercles were also present in the kidney, pericardium, and peritoneum. The mesenteric and bronchial glands were enormously enlarged, and in many places caseous. Tubercle bacilli were present in all the lesions. None of the lesions in the internal organs were of syphilitic origin.

Case II.—Victoria S., twenty-four days old. The mother had previously brought three children suffering from congenital syphilis to the same clinic. She had previously stated that she had not had syphilis. Nothing was known of the father, and it is not certain that either parent was tuberculous. The child had snuffles and presented all the characteristic appearances of congenital syphilis. There was a confluent papular syphilitic eruption on the nates and elsewhere. The percussion note over the left lung was dull and the breathing bronchial, with consonating râles. The spleen was slightly, and the liver markedly, enlarged. The temperature was normal. Mercurial treatment was ordered.

The patient was shown at the Vienna Dermatological Society as a case of syphilitic pemphigus and syphilitic pneumonia. The child died on the thirty-eighth day.

On post-mortem examination the left lower lobe was solid from greyish-white infiltration. There was acute catarrh of the bronchi of the left upper lobe and throughout the right lung, also of the larynx and trachea. The mediastinal and bronchial glands were enlarged, but not obviously caseous. The liver was large, reddish brown, somewhat indurated and with rounded margin. On microscopical examination of the lungs confluent peribronchial and perivascular tuberculosis was found, with tubercle bacilli. In the liver recent interstitial inflammation was present, with fatty degeneration of the liver cells. No trace of tuberculous lesions were found in any other organ than the lungs.

Case III.—Auguste G., eleven weeks old. The mother, æt. 28, was suffering from pulmonary tuberculosis. She had had five illegitimate children, and denied having had syphilis. Nothing was known of the father. The child presented the characteristic appearances of congenital syphilis, and had snuffles and a syphilitic rash on the buttocks. The rash appeared during the third week. The child had suffered from cough since it was five weeks old. There was doubtful dulness over the right lower lobe, with bronchial breathing and abundant moist râles. The liver was very large and hard, with rounded edge. The spleen extended four fingers' breadth below the costal margin. The temperature was normal. Mercurial inunction was ordered. The child died aged sixteen weeks.

Post-mortem.—The right lower lobe was solid from a homogeneous, greyish-white infiltration. Greyish-red and yellow tubercles were disseminated throughout the upper lobe. The lower half of the left lower lobe was collapsed. The bronchial glands were enlarged and caseation was commencing. The liver was fatty and slightly granular. In the portal fissure there was a caseous lymphatic gland the size of a hazel nut. The mesenteric glands were caseous. The spleen contained a large caseous nodule. Microscopic examination showed the characteristic signs of 'chronic tuberculous broncho-pneumonia, tuberculosis of the spleen and mesenteric glands, and syphilitic interstitial inflammation of the liver, with well-developed inflammation of the vessels.'

It is clear from the perusal of these cases that it will be necessary in future, even when the evidence of syphilis in the fœtus is undoubted, to examine carefully for tubercle bacilli before a pulmonary lesion is attributed to syphilis.

Acquired syphilis.—To present a trustworthy account of the morbid anatomy of acquired syphilis of the lungs is a far more difficult task than that just attempted. In considering a matter of such uncertainty we have preferred to rely upon evidence which is at hand and may be put to the test, rather than upon that to be found in the literature of a period when, owing to the absence of any certain test for tuberculosis, the difficulty of distinguishing between the pulmonary lesions of tubercle and syphilis was almost insuperable.

A study of the specimens of pulmonary syphilis contained in the London museums shows that the possibility of the changes being due to tubercle was in nearly all cases carefully considered. These specimens and the records connected with them probably constitute the most trustworthy evidence on which to base a description of the morbid anatomy of the acquired disease, and, as will be seen hereafter, they have been fully utilised.

Pathology and morbid anatomy.—Bronchial catarrh may occur as a manifestation of the secondary stage of syphilis, and possibly also of the period of incubation (Walshe). The fact that bronchitis, occurring without obvious cause in syphilitic subjects, may be greatly alleviated or cured by the administration of mercury is strongly in favour of this view. In the late secondary and tertiary stages gummatous infiltration of the submucous tissue of the trachea and bronchi is not infrequent, and may be followed by the formation of fibrous tissue which, subsequently undergoing cicatrisation, produces stenosis, one of the most characteristic syphilitic lesions in the main bronchi.

No definite statement can be made as to the most common period of the occurrence of gumma in the lungs; cases of which the real nature could not be doubted have been recorded as early as two years and as late as twenty years after infection.

The pulmonary lesions of acquired syphilis belong chiefly to the late tertiary stage of that disease.

A. **Gumma.**—Gummata may occur either singly or in numbers, and may vary in size from that of a hempseed or a hazel nut to that of a hen's egg, but the latter size is of rare occurrence. A gumma may be found in any part of the lung, but more commonly within its substance than upon the surface; and more often about the root, near the large vessels and bronchi, than elsewhere. The lower lobes are perhaps more often affected than the upper.

A gumma is rarely seen in the very early stage, of which alone the name is in any sense descriptive; but it is said then to present a gelatinous or glutinous appearance, thus resembling a similar growth in the liver. At a later stage it is of a grey colour, tinged with various shades of red, white, or yellow, and presents on section a smooth and semi-transparent appearance. At a still later period a gumma forms a well-defined nodule of a yellowish colour, firm and dry. Inflammatory changes in the surrounding lung may lead to the production of a well-marked fibrous capsule, but this may be absent. The gumma may break down, and its contents having been discharged, an irregular cavity may result; but this is, both absolutely and also in comparison with the occurrence of a similar change in caseous tubercular masses, very rare.

The chief difference between a gumma of the skin, for example, and one of the lung, is that whilst the former tends towards necrosis, the latter tends to be transformed into a mass of scar tissue, the contraction of which causes puckering of the surrounding lung and overlying pleura. By the deposition of lime salts a gumma may become calcareous.

In histological structure a gumma of the lung does not differ essentially from a similar growth elsewhere. In the early stage it is seen to consist of a granulation tissue composed of small cells about $\frac{1}{2500}$ inch in diameter, arranged concentrically around the sheath of the small vessels, and in some cases around the small bronchi. At a later stage the nodule becomes opaque in the centre, and its cellular structure can no longer be recognised; but fatty and albuminous granules are seen instead in the meshes of a dense fibrous stroma. Finally it becomes converted into a mass of dense cicatricial tissue. A gumma may form a centre from which a small-celled growth may infiltrate the surrounding tissue, spreading chiefly along the bronchioles.

The walls of the neighbouring alveoli are also infiltrated with small cells; and the alveolar spaces contain inflammatory products, due either to epithelial proliferation or to the presence of cells of a character similar to those constituting the nodular masses already described. Giant cells are occasionally present, but are not so characteristic a feature of gumma as of tubercle.

According to Dr. Councilman[1] the essential process in the production of a gumma in the lung is a pneumonia with fibrinous exudation, accompanied by fibrous change in the alveolar walls, the whole subsequently undergoing caseation. The first step in the process is stated to be a hyaline degeneration of the capillaries of the affected area ; this is followed by atrophy of the alveolar walls. The alveoli become distended with large pale epithelial cells and fibrin ; the cells also undergo the hyaline degeneration, forming smooth bodies staining with eosin, and varying in size from one-half the diameter of a red-blood corpuscle up to that of a large epithelial cell. The capillaries become converted into rigid tubes, and their lumen is much narrowed. Similar changes occur in the small veins and arteries. Immediately around the bronchi and arteries there is a formation of connective tissue, and here the alveolar walls show much thickening and contain many small round cells.

The whole of the structures thus altered tend to undergo necrosis, and when that change is complete a caseous-looking mass results.

The following descriptions of specimens in the Museum of Guy's Hospital well illustrate the appearances presented by gummata in the lungs. It will be observed that all the specimens here described were removed from the lungs of adults.

No. 254.—A portion of the upper lobe of a lung showing on the cut surface two masses, one of which was described in the recent state as 'consisting of a circumscribed nodule of a firm, yellowish, dry substance, corresponding in all particulars to that in the liver (a gumma), except in being somewhat less firm : the other is softening, breaking up, and in process of forming a cavity. Histologically the nodules are seen to consist of fibroid tissue, with many areas of caseation and a few giant cells.'

From the report of this case by Dr. Wilks the following additional particulars have been derived : The patient was a sailor æt. 29. No history was obtained ; he was moribund from laryngeal obstruction when admitted, and there was profuse expectoration of mucus and blood. There was a scar in the groin, and phimosis from a contracting sore on the penis. The whole mucous membrane of the larynx and trachea was deeply ulcerated, and the walls thickened by an infiltration of fibrous tissue into the submucous structure, producing great induration. The thyroid cartilage was bare at one spot, the lymphatic glands in the neck were enlarged.

The liver contained a dozen hard, round, fibrous tumours—the largest the size of a marble—yellowish white, tough, and of leathery consistence, dry, and emitting no juice on pressure. In two or three the circumference of the tumour consisted of a translucent structure ; and this was evidently the more recent formation, the opaque and yellow parts being probably tissue undergoing a degenerative change. At one spot a deep cicatricial appearance was produced by the contraction of a group of these small nodules.

---

[1] Councilman, *Johns Hopkins Hospital Bulletin*, vol. ii. No. 11, 1891.

Microscopically the nodules consisted of nucleated fibres and fibrous tissue.

No. 255.—The lower lobe of a left lung from a male patient æt. 27, who died from erysipelas of the larynx. The specimen shows at its hinder part a large yellowish mass partially separated from the surrounding tissue. Smaller nodules are seen in the adjacent lung. The pleura over the gumma is much thickened. Histologically the nodule consists of fibrous tissue which stains with difficulty. There were many gummata in the liver. With the exception of the above lesions and some bronchitis the lungs were healthy. There was a chancre on the penis, and suppurating buboes.

No. 256.—A portion of lung showing scattered through it several small masses of irregular shape, yellowish in colour, and firm on section. These masses are easily separable from the surrounding lung, which is healthy. Histologically the nodules show a central area of caseous material surrounded by a narrow zone of fibrous tissue, in which are many small round cells. The liver contained a single gumma, and was in a condition of diffuse syphilitic hepatitis. There were several gummata in the testes. From a male patient æt. 39, who had suffered from cough and dyspnœa for six months. He was admitted for hepatic ascites and slight jaundice.

The following specimen from the same Museum illustrates the appearances presented by a gumma which has undergone fibrous transformation :

No. 253.—A section of a right lung. From a man æt. 36, admitted for fracture of the cervical spine. At the upper part of the lower lobe is a circumscribed patch of fibroid material, with radiating processes extending into the surrounding pulmonary tissue. The pleura over it is much thickened. The interlobar septum is thickened, and from its upper portion similar fine fibrous strands radiate into the upper lobe. Other portions are very emphysematous (also fibroid and pigmented). No tubercle was found anywhere. There was lardaceous disease of the liver, spleen, and kidneys. Both testes were good specimens of syphilitic orchitis.

The following description of a specimen in the Museum of St. George's Hospital illustrates a combination of the caseous and fibrous stages of a gumma : [1]

'Section of a right lung near the root. In the posterior and upper part of the lower lobe, close to the spine, there is an area showing marked fibrosis ; situated within it is a caseous mass the size of a marble, somewhat loose. The overlying pleura is adherent and thickened ; bands of thick greyish fibrous tissue pass inwards from the pleura, and joining with each other form a meshwork.' No tubercle in any organ; surface of the liver scarred from perihepatitis. Large caseous gumma near the portal fissure, with smaller ones in its neighbourhood. Liver cirrhotic and lardaceous. Gummata in both testes. From a male patient who contracted syphilis in 1884, six years previous to his death. In 1886 he suffered from syphilitic disease of the testes and sores on the right elbow. Death was due to uræmia.

---

[1] Rolleston, H. D., *Path. Soc. Trans.*, xlii. 50.

B. **Lobular or broncho-pneumonia.**—A careful review of the evidence on which it is believed that inflammatory changes of the lobular or broncho-pneumonic type occur as the direct result of syphilis impresses one with the conviction that many of the cases described in the past as presenting such lesions were really cases of tuberculosis.

In the following case,[1] however, such a possibility may be excluded. It will be observed that the pulmonary lesions were secondary to and in continuity with the growth of large gummata in the liver and spleen. The specimen is in St. George's Hospital Museum.

Left lung.—The lower lobe is deeply congested and partially consolidated; the consolidation is in patches as in catarrhal pneumonia. Some of these masses appeared purulent, others fatty or caseous. The size varied from 3 mm. to ½ mm.; each patch or nodule was surrounded by a deeply congested zone. *Right lung.*—The lower lobe presented changes similar to the above; it was adherent to the diaphragm, through which a large caseous gumma in the liver had extended into the lung. At the upper margin of the caseous mass there was much fibrous induration and exudative consolidation of the pulmonary tissue. For the microscopical changes, which are given in great detail, the reader is referred to the original article. There was a gummatous mass chiefly in the upper part of the right lobe of the liver measuring 5½ in. by 4¼ in., and another occupying the upper third of the spleen. That organ was greatly enlarged, weighing 2 lbs. 6 oz. Both liver and spleen were firmly adherent to the diaphragm, and the muscular tissue of the latter was in part destroyed by the extension through it of the gumma in the liver. The specimen was taken from a man æt. 43, who contracted syphilis in 1861, twenty-five years before his death. He had periostitis of the tibia in 1864, left hemiplegia in 1871, and again in 1876.

C. **Fibroid induration.**—The following are the more important changes of this nature which have been attributed to syphilis: (*a*) thickening extending from the hilus around the bronchi and vessels; (*b*) isolated masses of fibroid tissue in various parts of the lung; (*c*) diffuse changes occupying the whole or the greater part of one lung.

The marked tendency of gummatous lesions to spread along the vessels and bronchi has already been referred to.

---

[1] Delépine and Sisley, *Path. Soc. Trans.*, xlii. 141.

The following case [1] is an example of syphilitic fibrosis illustrating the first variety of this lesion :

Woman æt. 50.—Thrombosis of cerebral artery ; hemiplegia. Pigmented excavated scars on left leg, due to old syphilitic ulceration. Emphysema. *Lungs.*—Right lower lobe contained a deep depression and a much-puckered cicatrix due to pigmented fibroid bands running into the lung tissue. No caseous or calcareous nodules. No pleural adhesions. *Microscopical examination.*—The fibroid tissue is arranged chiefly around the vessels and bronchi with a more or less concentric disposition. The coats of the vessels are much thickened. There is a small-celled growth invading the alveolar walls, which are also much thickened. In places the cells and nuclei are aggregated in heaps.

As an example of fibrosis in the form of scattered areas of induration, the following case may be cited from the same source :

Woman æt. 25.—Fracture of cervical spine. Pigmented and puckered cicatrix and syphilitic ulcers on left leg. Calcified gumma in the liver. *Right lung.*—Upper lobe healthy. Middle lobe presented in the centre large irregular patches formed by radiating bands of fibroid tissue ; also smaller scattered patches of the same nature ; the band whitish, not pigmented. One patch contains a calcified nodule. No pleural adhesions. *Left lung.*—Adhesions over lower lobe ; and whitish, puckered, depressed fibroid patches with irregular thickening of the pleura. On section extensive fibroid infiltration ; bands appear to run into the lungs from the pleura. Some small rounded caseous patches are also present.

The following specimen from the Museum of Guy's Hospital [2] illustrates the appearances met with in 'diffuse syphilitic fibrosis of the lungs.' The patient was a man æt. 54, who had suffered from winter cough for some years.

No. 252.—A portion of a right lung in which there is a considerable excess of fibroid material appearing on the cut surface as a delicate network traversing the pulmonary tissue in all directions. The fibroid change is less marked at the apex than at the base, in which latter situation many of the air vesicles are dilated ; over this area the pleura is slightly thickened and is adherent. The dense fibroid tissue that pervades the lung shows, scattered through it, numerous collections of small round cells not undergoing caseation. No giant cells are present. The walls of the small arteries are thickened. One or two small cavities the size of peas, with soft caseous contents, were situated near the root of the right lung, probably softening gummata ; no tubercle bacilli could be found in them. The condition of the left lung resembled that of the right. The liver was scarred ; the testes were fibroid. Death was due to bronchitis.

D. **Changes in the bronchial glands and lymphatics of the lung.**—In a case of syphilitic disease of the liver, lungs, dura mater, cranium, and sternum recorded by Dr. Hermann Weber,[1] the bronchial glands and lymphatics of the lung presented the following appearances: The bronchial glands were much enlarged—some being the size of a pigeon's egg, some only that of a hazel nut. From the greyish-white section of the larger glands, which were rather soft, a creamy fluid exuded, consisting of fat globules, granular corpuscles, and an abundance of large cells in a condition of fatty degeneration. The less enlarged glands were harder, their sections offered a marbled appearance, large white patches, almost like bacon, being interspersed with greyish-red, very vascular tissue. No juice exuded spontaneously or could be squeezed from the section. Large nuclei and nucleated cells were the principal microscopical elements, with a very small proportion of fibres thickly studded with nuclei. The lymphatics leading from the lungs to the enlarged glands were dilated and their ramifications on the surface and throughout the lungs were distended with creamy fluid.

---

[1] Greenfield, *Path. Soc. Trans.*, xxviii. 248.

[2] Perry, E. C., *ibid.* xlii. 53.

A similar appearance is described in the case of Drs. Delépine and Sisley already quoted. 'Immediately under the pleura there was a network composed of ramified tracks. The appearance suggested lymphatics distended with cells or some fatty products.' The lymphatics of the subserous layer of the pleura were considerably enlarged over areas corresponding to the yellow patches (? of syphilitic broncho-pneumonia) within the lung.

Dr. Weber rejects the view that the bronchial glands were first affected by the syphilitic virus, and that the engorgement of the pulmonary lymphatics resulted from obstruction to the passage of the lymph.

E. A progressive destructive disease, the so-called 'syphilitic phthisis.'

It appears to the writer that the question of the existence of a syphilitic lesion of the above nature can only be settled by a careful study of cases which fulfil the following conditions:

(i) The cases must be complete—that is, the symptoms observed during life must be considered in connection with the lesions discovered on post-mortem examination.

(ii) The evidence of syphilitic infection must be undoubted.

(iii) Repeated examinations of the sputum must have been made, and tubercle bacilli have been invariably absent, and the absence of tubercle from the lungs (as the cause of the lesions) must be proved by post-mortem examination.

(iv) Syphilitic lesions about the nature of which there can be no doubt must be found in other organs.

It is from such evidence alone that we can hope to construct the clinical history and morbid anatomy of advanced syphilitic disease of the lungs.

The following cases illustrate this variety of the disease:

Case I.—Charles N., æt. 38, bricklayer. In 1892 he suffered from cough, with expectoration and pain on the left side of the chest. In 1893 he had night sweats and dyspnœa. He has not lost weight, and, beyond an occasional streak of blood in the sputum, there has been no hæmoptysis. From January to May 1894 he was an in-patient of the Brompton Hospital, under the care of Dr. Mitchell Bruce; the diagnosis then recorded was 'Syphilis (?), tracheal stenosis, chronic bronchial catarrh, induration of the left upper lobe and of the left base with pleural adhesions over that area. Cicatrisation of the soft palate and adhesion of the right posterior pillar of the fauces to the back of the pharynx.' There were no bacilli in the sputum. He continued fairly well until October 1894, when he expectorated a large quantity of offensive purulent material for two consecutive days. Cough was very severe at this period. His health subsequently improved, and remained so until February 20, 1895; when in the course of a few days he brought up about a quart of blood-stained sputum. Hæmorrhage then ceased and dyspnœa diminished. On March 5 cough and dyspnœa increased and he became seriously ill, with constant headache and slight delirium. Œdema of the feet subsequently supervened. On March 15, 1895, he was admitted to the Brompton Hospital under the care of Dr. Percy Kidd. On admission he was reported to be fairly well nourished; stridor marked; cough severe. Right lung resonant everywhere; breath sounds much exaggerated, expiration prolonged. Loud hoarse inspiratory and expiratory stridor all over the lung; sibilant rhonchi general. *Left lung.*—Expansion much diminished; resonance much impaired, front and back; breath sounds weak; expiration prolonged; fine crackling râles over the whole of lung; vocal fremitus and resonance diminished. Expectoration profuse and difficult to expel. No tubercle bacilli found. Temperature 99° F. It varied between that point and 96° F. during the time the patient was in hospital. The dyspnœa gradually increased and death occurred on April 10.

Post-mortem.—Scars on tongue, glans penis, and scrotum; and adhesion of skin to left testis. Marked thickening of the right tibia. Larynx normal. Trachea narrowed at the lower end. Recent ulceration from cricoid downwards for two inches; below this, down to point of bifurcation, there was extensive scarring of the cartilaginous portion; and also at its line of junction with the posterior wall. The submucous tissue was extremely thickened. Cartilages bare in several places. The main bronchi were much scarred and showed extreme narrowing. The bronchus to the left upper lobe was impermeable to a probe. *Right lung.*—Old pleural adhesions over the upper lobe, recent pleurisy with effusion at the base. Emphysema, with reticular fibrosis especially around bronchioles. Deep in the upper lobe at the edge of one of the main bronchi there was a large black fibroid mass, with fibroid radiation into the surrounding tissue; in other parts two small, hard, raised masses, one with fibrous strands running up to it. Base solid from broncho-pneumonia. No appearance of tubercle

Left lung.—Upper lobe extremely contracted, containing no normal tissue. It consisted of deeply pigmented blackish-grey fibrous

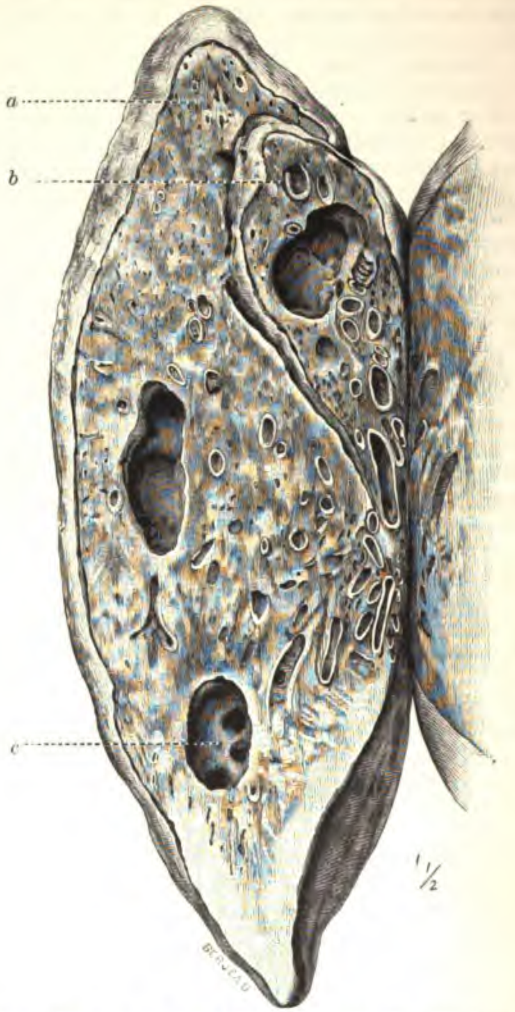


FIG. 113.—CHANGES IN THE LUNG SECONDARY TO SYPHILITIC STENOSIS OF THE TRACHEA AND MAIN BRONCHI

a, compensatory enlargement of lower lobe, which forms the apex of the lung; *b*, the contracted left upper lobe; *c*, cavity in the lower lobe, with dilated bronchi in its neighbourhood. The changes in trachea and bronchi in this case are shown in fig. 46, p. 78.

tissue surrounding the openings of bronchial tubes, and bronchiectasis. At the centre there was a smooth-walled cavity about the size of a small chestnut into which a bronchus opened. No appearance of tubercle. Lower lobe.—Emphysematous, with reticular fibrosis along the margin and at the base. Bronchi dilated, but not to a marked degree. About the centre point of the outer margin there was a small nodule, probably a gumma, white and firm, and surrounded by a pigmented fibrous capsule. The extreme base consisted of indurated fibrous tissue extending from the pleura to a cavity, the size of a marble, into which a small bronchus opened. From this cavity fine fibrous bands radiated in all directions, producing extensive fibrosis of the surrounding lung. Perihepatic and splenic adhesions. Liver scarred and nutmeg. Spleen contained several calcareous masses surrounded by a fibrous capsule. Testes fibrous.

Case II.—R. D., æt. 36; coachman. The family history is unimportant. At the age of 18 he had a sore on the penis, for which he was treated for several months with medicine and a lotion. In 1890 he became an out-patient under the care of Dr. Fowler at the Brompton Hospital; he was suffering from cough and expectoration, which continued. There was an enlarged gland at the lower part of the neck on the right side, dulness at the right apex, with feeble breath sounds and bronchial breathing in the right supraspinous fossa. The liver was large, nodular, and very tender. He took iodide of potassium in gradually increasing doses and obtained some relief. He was subsequently an out-patient at the Middlesex Hospital. In March 1893 he caught a severe cold, but remained at work. In the following April he noticed œdema of the legs and scrotum. He was admitted into Middlesex Hospital, under Dr. Cayley, on May 13, 1893. He was pale and emaciated, the legs and scrotum were œdematous. He had troublesome cough, accompanied by the expectoration of large quantities of extremely fœtid pus. The breath was fœtid.

Physical signs.—Expansion deficient on right side. Relative dulness at right apex front and back, breath sounds feeble over dull area. Absolute dulness from level of fifth interspace in nipple line and in axilla to base; behind from angle of scapula to base. Vocal fremitus and resonance diminished, and breath sounds scarcely audible over dull area. Left side normal. No displacement of heart. Hepatic region prominent. Liver dulness extended three inches below the costal arch in right mammary line and almost to umbilicus in middle line. Liver somewhat soft and elastic.

Urine, sp. gr. 1·004, neutral, contained albumin and fatty casts. The expectoration consisted of frothy greenish pus, forming thick masses in a watery fluid. It contained no tubercle bacilli.

May 17.—The chest was explored in the axillary and submammary region. No pus was found. *May* 19.—Liver exposed by incision below costal arch, and a depressed cicatrix seen. The expectoration continued copious, green, and fœtid. Absolute dulness appeared over whole of right side up to clavicle, with amphoric breathing and pectoriloquy below the clavicle. *June* 15.—Offensive pus was evacuated through a cannula inserted in third right interspace in mid-axillary line: a portion of the fourth rib resected, lung incised, more pus evacuated, drainage tube inserted. *June* 19 *and* 20.—Hæmorrhage from wound. *June* 21.—Death.

Autopsy. Abstract of notes.—Old syphilitic scar in trachea, six rings above bifurcation, more recent scar at bifurcation, producing stenosis of the main bronchi to the right upper and lower lobes. One bronchial gland enlarged. Pleura over right lower lobe adherent and much thickened. Bronchi much dilated beyond the site of stenosis. At the base of the upper lobe were two large irregular cavities with sinuous outlines communicating with large bronchi, lined by a distinct membrane and containing sloughy portions of lung tissue. The anterior cavity had been opened by the incision. The section of the lung was smooth and presented a finely speckled yellow appearance. No pus exuded from the yellow spots on pressure. In the anterior part of the lower lobe there was a large irregular cavity, the walls of which showed no sign of any mucous membrane, they were covered with yellowish-grey sloughy material. No tubercle present and no caseation. The lung puckered in many places and fibrous almost throughout. Liver enlarged (76 oz.). Large puckered cicatrix on the upper surface of the left lobe, and many similar cicatrices elsewhere. A cretaceous and caseous gumma on the posterior aspect of the right lobe. Liver substance fatty and amyloid. Kidneys large, pale, amyloid, and fatty.

CASE III.—T. H., æt. 59; painter. Admitted into Middlesex Hospital under Dr. Fowler, February 4, 1893. Father died aged 70; mother aged 75. No history of tuberculosis in family. Accident to left knee æt. 19, followed by formation of an ulcer. Chancre on penis in 1858 (æt. 25), secondary rash and sore throat subsequently. In 1864 ulcers on left leg, and twice subsequently. In 1880 ulcer on right leg, near external malleolus. Dry cough since 1887, worse in winter. Since December 1892 severe paroxysmal cough with offensive mucopurulent expectoration. Marked emaciation during this period.

A pale, grey-haired, emaciated man. Breath very fœtid. Extensive scars on left leg of old standing, more recent scars on right leg. Scar in right lumbar region where incision was made for 'abscess.' *Right lung.*—Hyper-resonant on percussion; breath sounds at apex bronchial, front and back; crackling râles in supraspinous fossa. Bronchophony and pectoriloquy well marked in same area. Dulness over lower lobe to angle of scapula, breath sounds bronchial, with coarse crackling râles over same area. *Left lung.*—Resonance impaired over clavicle and in supraclavicular fossa, elsewhere hyper-resonance. Bronchial breathing over upper lobe, front and back, with crackling râles. Breath sounds bronchial over upper part of lower lobe, with bubbling and coarse crackling râles, the latter extending to the base. Urine, sp. gr. 1·020; no albumin. Expectoration copious, purulent, and offensive. Frequent examinations made for tubercle bacilli, but none found. No elastic tissue found. Temp. 98°, pulse 84, respirations 44. *February* 21.—

Dulness at both apices, and medium crackling râles. Temperature between 99° and 100° F. The respirations between 36 and 48. Cough severe, and the breath and expectoration offensive. Died February 23.

Abstract of post-mortem notes.—Scar on corona of penis with some induration around. Calvarium thickened, dura mater adherent. Pleural adhesions over both lungs. *Right lung.*—Emphysema along anterior margin and at base. Apex pigmented and consolidated from pneumonia and œdema. In lower part an oval cavity measuring 2½ inches by 2 inches, in communication with main bronchus, and containing greenish-yellow, offensive, shreddy material. Below this for an inch and a half the lung grey in colour and almost solid, a few small cavities with curdy contents. No tubercle found. The pleura covering the consolidated area much thickened. *Left lung.*—Upper lobe pigmented and 'nodular.' A cavity, from bronchial dilatation, occupies the posterior portion. The lower lobe emphysematous, and contained numerous encapsuled caseous masses about 2 mm. in diameter. Bronchial glands pigmented, but not caseated. No ulceration in air passages. No gummata in liver or spleen. Testes scarred and fibrous. Small white fibrous nodule in right kidney.

The following cases are incomplete, and do not attain to the standard of evidence laid down, inasmuch as the patients are believed to be still living.

Case IV.—Mary G., æt. 33, married. Three children alive, three dead—one stillborn, one died a few hours after birth. Has had four miscarriages. Admitted into the Brompton Hospital, June 13, 1894, under Dr. Fowler. No history of tubercular disease in the family. Ten years ago had some affection of the liver. Three years ago had an attack of influenza followed by pleurisy (R) and congestion of the lungs. Right pleurisy recurred in August 1893. Has had a slight cough for three years, worse since September 1893. Expectoration has been profuse, and for the last two months fœtid and of a bitter taste. In October 1893 it was tinged with blood for three weeks. Dyspnœa worse since September 1893. Catamenia ceased since the birth of the last child on September 30, 1893, at which time she caught a chill. In February 1893 patient noticed a swelling in the left loin, which at first gradually increased in size and subsequently diminished. It is slightly movable and is not tender. It is about equal in size to a small Tangerine orange, is situated rather superficially, and over the erector spinæ muscle, whether actually within the muscle could not be determined. Emaciation, cough, and weakness have been increasing lately, and night sweats have been continuous.

Physical signs.—*Right lung.* Marked flattening of the whole of the right side, particularly in front. Measurement at right nipple level: right 14½ inches, left 16 inches. Dulness over upper lobe, with distant cavernous breathing and bronchophony front and back. Impaired resonance over upper part of lower lobe posteriorly, where crackling râles are audible; similar râles at the right base where percussion note is dull. *Left lung.*—Harsh breathing general (? compensatory), no adventitious sounds. Liver much enlarged and nodular on the surface, margin irregular; extends from the fourth space to below the umbilicus. Spleen not enlarged. Urine free from albumin. Expectoration profuse and fœtid. No tubercle bacilli. From June to September the expectoration was usually fœtid. Bacilli repeatedly sought for, but never found. The cavity at the right apex extended. *September* 5.—Retraction more marked at right apex. Cavity dry. Numerous crackling râles in axilla, and all over base. General improvement. Liver appears more nodular. *October.*—Large crackling râles over base and in axilla. Cavity at apex dry. No bacilli to be found. Discharged October 13. Intratracheal injections of menthol appeared at first to have an effect in diminishing and then removing the odour of the expectoration; but subsequently the fœtor returned and appeared to be uninfluenced by their continued use. The quantity of expectoration was small during the period over which their administration extended; but it had been steadily diminishing up to the time when this treatment was commenced. The patient considered that she derived benefit from the injections. The nature of the tumour in the back was doubtful; it was believed to be a gumma in the superficial part of the muscle. Inunction of mercurial ointment was made daily into the back from September 22 onwards.

CASE V.—Edward C., æt. 47; a waiter. Admitted into St. George's Hospital, April 13, 1894, under Dr. Whipham.[1] His father and mother died of 'consumption.' Thirty years ago he had a hard chancre. He has had syphilitic psoriasis of the palms. He has not had hæmoptysis, night sweats, or emaciation. A fortnight before admission he was attacked with severe pain on the right side of the chest and dyspnœa. On admission he was anæmic, and complained of cough and profuse expectoration. The skin was of a brownish tint and presented numerous old rupial scars. *Right lung.*—Impaired resonance over upper lobe with feeble breathing. Just below the second rib there is a small area of increased dulness and cavernous breathing with whispering pectoriloquy. There are rhonchi all over the right lung and to a smaller extent over the left. The sputum is profuse and mucopurulent. No tubercle bacilli were found on any occasion, the examinations were made by several observers. *April* 15.—Ordered Potiodid. gr. v., Liq. hydrarg. perchlor. ʒj. ter die. *April* 28.—Expectoration and cough less. Physical signs at right apex less marked. *May* 2.—Discharged to convalescent home.

The following case illustrates the fact, first pointed out by Dr. Pearson Irvine,[2] that stenosis of a main bronchus may give rise to destructive changes in the lung. It will be observed that the case was one in which a recent tuberculosis supervened on old syphilitic disease.

---

[1] The writer is indebted to Dr. Whipham for his kind permission to use the notes of this case.

[2] *Path. Soc. Trans.*, xxviii. 30.

CASE VI.—Margaret S., æt. 25. Admitted into the Brompton Hospital June 25, 1884, under Dr. Reginald Thompson. Family history good. Good health up to two years ago, when after marriage she had 'ulcerated legs.' No sore throat or skin eruption. Cough, expectoration, pain in left side, dyspnœa, night sweats, and emaciation have been present for eight months. On admission the fingers were clubbed; there was a large circular ulcer on the back of the left thigh with some scarring, and coppery staining about the knee and leg on the same side. Cough, more or less paroxysmal; expectoration copious, nummular, and purulent. No tubercle bacilli. Right chest 16½ inches, left 15½. Dulness over left lung, absolute at base, where vocal fremitus is absent; elsewhere it is diminished. Bronchial breathing, pectoriloquy, and crepitation over left upper lobe. Breath sounds absent at base, some rhonchus there. Slight crepitation at right base.

The ulcer on the thigh yielded to antisyphilitic treatment. The physical signs remained much the same except that the breath sound at the left apex became cavernous. There was well-marked pyrexia throughout. The expectoration remained copious, at times it averaged a pint in the twenty-four hours. Death occurred on March 1, 1885, and was preceded by anasarca, ascites, and profuse diarrhœa.

Autopsy.—A few small scars in the subglottic portion of the larynx. The lower half of the trachea marked by numerous stellate puckered cicatrices, involving both membranous and cartilaginous portions, but especially the latter. The origin of the left bronchus represented by a small opening just admitting a probe; the surrounding parts of the tracheal wall extremely fibrous and puckered. Slight scarring in the right bronchus about the origin of the upper lobar branch. Left lung excavated from apex to base. Numerous trabeculated cavities in the upper lobe intersected by tough pigmented bands; walls thin and smooth. The cavities larger behind than in front, in the latter region they were more numerous; and the intervening fibroid induration was more pronounced. Some bronchi appeared to expand uninterruptedly into the smaller cavities. Numerous small cavities in the lower lobe situated in indurated fibroid lung. The cavities contained extremely fœtid reddish fluid secretion, and in some places some soft putty-like material. No tubercular nodules in this lung. The contents of the pulmonary cavities including the liquid and caseous parts were carefully examined for tubercle bacilli, but none could be found. Right lung crepitant, but studded with large tubercular groups, which were most plentiful in the middle lobe and lower part of the lower lobe. Amyloid disease of thyroid, mesenteric, and mediastinal glands, also of the kidneys, liver and spleen, and mucous membranes throughout the body.

The recent tuberculosis of the right lung was obviously quite unconnected with the disease in the left, which was secondary to the bronchial stenosis.

This case proves very clearly that a progressive destructive disease of the lung may result from syphilitic stenosis of a main bronchus; but it does not prove that this disintegration of the lung is due to the continued action of the specific virus of the disease, as is the case in pulmonary tuberculosis. The fact that lesions similar to those here described may occur when the narrowing of the bronchus is due to pressure from without, as by an aneurysm, shows that the bronchial obstruction is the main factor in their production. Stenosis of the bronchus is followed by retention of secretion in the tubes, and this by bronchiectasis. Decomposition of the retained secretion induces inflammatory changes in the surrounding lung, and finally the part so affected breaks down and cavities are formed.

The cases here described prove that in individuals undoubtedly the subjects of syphilis widely-spread destructive changes may be found in the lungs; and that such lesions may occur independently of the presence of tubercle. Whether they are such as to entitle the condition to be termed 'syphilitic phthisis' must be decided by those who continue to use the word 'phthisis,' a term which many teachers have ceased to employ.

If the name 'phthisis' is given to a group of symptoms and morbid changes, it can hardly be denied that a case (see Case I.) which is marked by such symptoms as severe cough, dyspnœa, emaciation, fever, night sweats, profuse expectoration, and hæmorrhage, and which, on examination after death, is found to present signs of consolidation, fibrosis, and excavation of the lungs, belongs to that category. The task before us, however, is to determine the real nature of the pathological lesions of pulmonary syphilis and of the symptoms which they produce; whether they are such as to warrant the use of a vague nomenclature which it would be well to discard is a question of but little importance. It may be of service, however, to draw attention to the chief points of difference between the pulmonary lesions of tuberculosis and syphilis.

I. Tubercle usually affects the apex of the lung and subsequently the apex of the lower lobe, and tends to progress along a certain route. The primary lesion of syphilis is often about the root and central part of the lung; the disease follows no definite line of march, and gummata may be found in any position.

II. Both tubercles and gumma may undergo either necrosis and caseation, or fibrous transformation; but with caseous tubercle the tendency towards softening and cavity formation is the rule, whereas a caseous gumma very rarely breaks down.

III. The progressive destruction of the lung by a process of disintegration leading to a gradual increase in the size of a cavity, a change so commonly observed in tubercular disease, is rarely if ever observed in syphilis, except as a secondary result of stenosis of one of the main bronchi.

IV. In nearly all cases of advanced destruction of the lung occurring in the subjects of syphilis, stenosis either of the trachea or of one of the main bronchi is present, whereas this lesion is very rare indeed in tuberculosis.

V. The cavities found in cases of pulmonary syphilis are usually bronchiectatic, but not invariably so; whereas in tuberculosis they are commonly due to progressive destruction of the lung, but may be bronchiectatic.

VI. The tendency to the formation of pulmonary aneurysms, which is so marked a feature in tuberculosis, is rarely observed in pulmonary syphilis.

VII. Pulmonary lesions in tuberculosis are very common, whereas in syphilis they are extremely rare.

The necessity for prolonged specific treatment is certainly more generally appreciated now than formerly; and it is therefore probable that rare as these lesions have been in the past, they will be still rarer in the future. The conditions which favour their development are the neglect of mercurial treatment shortly after infection, and anything which, by lowering the general health, tends to diminish the resisting power of the individual.

When our knowledge of the virus of syphilis is as complete as that we even now possess of the bacillus tuberculosis, it may be possible to state definitely whether the destructive pulmonary lesions found in advanced cases of the acquired disease are directly due to the continued action of a specific micro-organism; at present the question remains unanswered.

Symptoms.—The only point worthy of mention in respect of syphilitic lesions of the bronchi is that the catarrhal signs which accompany the secondary stage are, as a rule, general in their distribution; whilst in the tertiary stage they are more often localised, owing to the tendency at that period to the formation of gummata in the main bronchi. Should stenosis occur there may at first be bronchial breathing limited in area, and often most marked about the root of the lung posteriorly. As the lumen of the tube diminishes, the breath sounds over the pulmonary area which it supplies become more and more feeble, and finally disappear when air ceases to pass the obstruction. If bronchiectasis is forming behind the site of stenosis there may be cough with profuse, purulent, and fœtid expectoration, accompanied by general signs such as emaciation and moderate pyrexia.

In the cases described in this chapter it will be observed that *cough* was as a rule the earliest and most prominent symptom. In the early stage it may be due to irritation, the result of laryngeal, tracheal, or bronchial lesions; at a later period it is probably chiefly due to the changes within the lung itself.

Dyspnœa comes next in point of frequency. It varies in severity with the nature of the lesion: slight when this is limited, in cases of extensive fibrosis or stenosis of one of the main bronchi, it may be very severe. The dyspnœa tends to become paroxysmal and to assume the characters of bronchial asthma. *Hæmoptysis* has not been of frequent occurrence in cases observed by the writer, but it may occur and may prove fatal. In one case of syphilis of the bronchial glands profuse and fatal hæmorrhage occurred from softening of a gland and its rupture into a main branch of the pulmonary artery.

Expectoration may be profuse, purulent, and offensive. Fœtor of the expectoration is common in cases of advanced pulmonary syphilis. The sputum will be free from tubercle bacilli.

Pain may be present, but is not a very prominent feature of the disease.

Emaciation is not as a rule nearly so extreme as in tuberculosis, but with advanced lesions in the lungs the difference is not so remarkable as to be of any value from a diagnostic point of view.

Night sweats were present in several of the cases here described.

When extensive lesions are present *pyrexia* may be considerable, and of the hectic type commonly observed in tubercular disease of the lungs, but in the early stages there may be a complete absence of fever.

The general symptoms, as will be seen on reference to the cases described, do not, in the presence of widely-spread lesions, differ markedly from those of advanced tubercular disease of the lung.

Physical signs and diagnosis.—The lesions of syphilis are rarely of such a nature as to produce signs by which they can be distinguished from others of an entirely different origin.

Consolidation and excavation will be recognised by their ordinary signs, probably before their syphilitic origin is suspected; and it appears therefore unnecessary to describe them in detail, more particularly as in the cases here recorded the results of the physical examination are given in full.

The features of pulmonary syphilis are certainly not as yet so clear that the disease can be recognised by any positive signs, but by a process of exclusion a diagnosis may generally be made.

The case will probably be regarded at first as one of pulmonary tuberculosis; but repeated examination of the sputum, and the failure to discover the presence of tubercle bacilli, will suggest another diagnosis.

A careful inquiry, possibly previously omitted, will now be made as to syphilitic infection and as to the occurrence of any secondary or tertiary manifestations. The absence of such a history in a hospital patient will not exclude syphilis; but it is rare in private practice for a patient to have had syphilis with tertiary symptoms and to be ignorant of the fact.

Evidence of tertiary lesions in the larynx, liver, spleen, or testes is of importance as showing that the viscera are affected.

Careful search should also be made for lesions of the calvarium, of the dura mater, and of the sternum and ribs.

Speaking generally, the diagnosis of pulmonary syphilis from tuberculosis will depend far more upon the examination of the sputum than on the results of physical examination.

A careful examination of undoubted specimens of pulmonary syphilis does not bear out the statement that the lesions are generally limited to the middle part of the lung; they are so often found elsewhere that little importance attaches to their exact site in deciding the question of diagnosis. It would be rash indeed to diagnose pulmonary syphilis because of a lesion situated in and apparently limited to the middle of one lung, without having previously demonstrated, by frequent examinations, the absence of tubercle bacilli from the expectoration. Such points, however, are not without importance, as they may serve to arrest attention, as being unusual in a case possibly hitherto regarded as one of 'phthisis' or 'consumption.'

Evidence of excavation with fœtid expectoration, which does not contain tubercle bacilli, should always suggest the possibility of the case being one of pulmonary syphilis. When the physical signs indicate stenosis of the trachea or of one of the main bronchi, and the presence of a growth or an aneurysm can be excluded, it is very probable indeed that syphilis is the main factor in the case.

Those who are content with a diagnosis of 'phthisis' and neglect the systematic examination of the sputum, will almost certainly overlook a case of pulmonary syphilis if it should come in their way.

A striking example of this has recently come under our notice. A military officer who had contracted syphilis some years back began to suffer from symptoms of laryngitis, and on examination of the chest, well-marked signs of disease were found at the apex of the right lung. The laryngoscopic appearances did not suggest to several competent observers that the lesion was due to syphilis, and the case was regarded as one of 'consumption of the throat and lungs.' It occurred to a medical man who saw the patient at a later period to examine the sputa for tubercle bacilli, and, as none were found on repeated examination, doubt was cast upon the diagnosis of 'phthisis,' and mercury and large doses of iodide of potassium were prescribed and the patient rapidly improved, but the stenosis of the larynx remained.

Prognosis.—Extensive pulmonary lesions, particularly excavation, whether from bronchiectasis or disintegration, and fœtid expectoration, are certainly very grave complications of syphilis. If there is also evidence of gummatous hepatitis, albuminuria, and amyloid disease, recovery is scarcely possible, and life is not likely to be much prolonged.

It is probable, however, that with our present improved means of diagnosis of tuberculosis of the lungs syphilitic cases which would have formerly been considered tuberculous may be recognised at an earlier stage than formerly, and may recover under appropriate treatment. In an undoubted case of pulmonary syphilis which came under the writer's care at a late period of the disease, the affection had been kept in check for many years by repeated visits to Aix-la-Chapelle, and by the active employment of antisyphilitic treatment. In any case seen in an early stage great improvement, if not complete cure, may reasonably be looked for from the use of similar measures. There are, however, limits to the action even of specific remedies; and it is not to be expected that lesions, such as bronchial stenosis and dilatation, extensive fibrosis and excavation, or gummata in a state of fibrosis, will disappear under the administration of mercury or iodide of potassium.

Treatment.—If the disease in the bronchi or lungs is recognised when in an early stage, the patient should be advised to undergo a prolonged course of treatment with mercury. Iodide of potassium in gradually increasing doses is generally administered at the same time.

If, however, the disease is advanced and the patient emaciated, it is better first to try the effect of iodide of potassium alone; giving at the same time cod liver oil and tonics. To maintain and improve the strength and general nutrition of the patient are matters of as much importance in the treatment of syphilitic as of tubercular disease of the lungs, and are to be secured by the same means.

The warm sulphur baths of Aix-la-Chapelle in association with mercurial inunction enjoy a special reputation in the treatment of syphilis, and are to be recommended to sufferers from pulmonary syphilis who are able to go abroad for treatment.

When tubercular disease of the lungs occurs in a syphilitic subject, the treatment will be mainly such as is suited to cases of tuberculosis. A mercurial course is rarely admissible, but iodine, in the form of the syrup of the iodide of iron, may be given with advantage.

In cases accompanied by fœtid expectoration, creasote vapour baths and intratracheal injections of guaiacol should be tried.

Cases of syphilitic disease of the lung accompanied by bronchiectasis have not, in the experience of the writer, been benefited by surgical measures undertaken with a view to drain the cavities.

J. K. F.

CHAPTER XXXVIII

ACTINOMYCOSIS

PAGE
Symptoms and diagnosis 453
Course. 456
Treatment 457

The recognition of actinomycosis as a disease in the human being dates only from the year 1878, though it had been described by Bollinger in the previous year as occurring in cattle, and had been figured as long ago as 1845 by Langenbeck in a case of abscess connected with vertebral caries, and also by Lebert in 1857 in a case of abscess in the chest wall. It is not proposed to enter fully into the history of this disease, for the details of which the reader is referred to what is now a very copious literature of the subject.

Symptoms and diagnosis.—For the present purpose it is only necessary to point out the symptoms of the disease as it affects the chest, and especially to indicate the difficulties involved in its recognition. These are undoubtedly great, partly because the symptoms are frequently very like those of pulmonary tuberculosis, or of disease of bone, or of tumour, but the principal reason why actinomycosis is so frequently unrecognised is because so few have seen the disease that the possibility of its occurrence is not present to the mind. We believe, however, that it is much more common than is usually supposed, and that, when it is more accurately described in the text-books, there will be fewer instances of its being overlooked than is now undoubtedly the case, in support of which it may be said that the occurrence of five cases of actinomycosis in a little more than a year in the practice of one of us can hardly be looked upon as altogether accidental.

It is known that the fungus makes its way into the body either by means of the mouth or through the alimentary or respiratory tracts. The affections of the mouth will not be considered here; but, should the entrance be effected through either of the other channels, the obvious symptoms may be chiefly thoracic; in such cases the disease affects either the lungs, the pleura, or the liver.

The history of a patient with this affection will probably be as follows: the onset of the disease will be gradual, the patient for some time complaining of nothing except weakness and gradual loss of strength ; there may be some cough setting in insidiously, with or without expectoration ; and this may or may not be followed by pleurisy, with or without effusion.

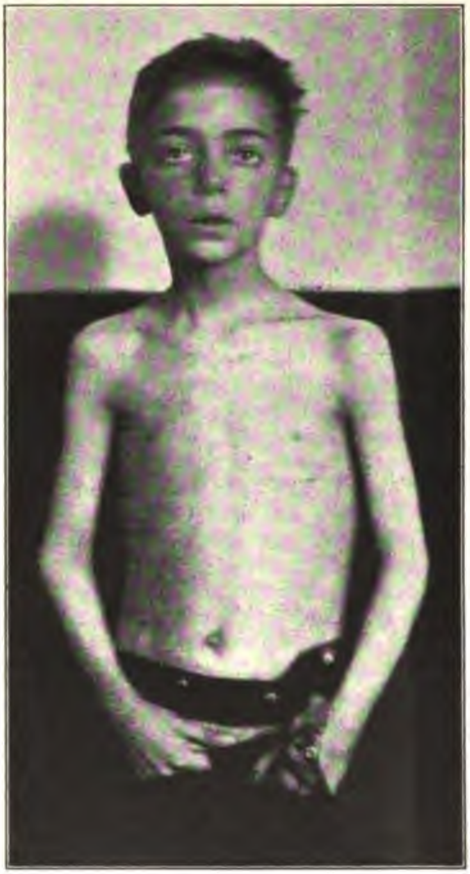


FIG. 114.—FRONT VIEW OF BOY SUFFERING FROM ACTINOMYCOSIS OF THE RIGHT LUNG AND PLEURA IN THE EARLY STAGE

The bulging of the mass is seen below and external to the right nipple.

We have no records of the *physical signs* in the early stages of the disease, nor do we know how soon any alteration in temperature manifests itself; but by the time a case comes under observation it is not unlikely that some or all of the following symptoms may be apparent.

The patient will be pale and anæmic, and will probably have lost flesh, and will complain of loss of appetite and of a troublesome cough. If there is expectoration it may be simple mucus or it may be purulent, and if so may possibly contain *yellow granules* which, if present and exhibiting the microscopical appearances described below, are characteristic of the disease. It is said that the sputa are sometimes rusty, as in pneumonia. It is also possible that the patient may say that he has expectorated at one time a large quantity of offensive yellow material. The temperature will be raised and may be of a hectic type. There may be localised dulness, or there may be the dulness characteristic of pleural effusion. Over the dull area it is likely that the breathing will be weak, and it is not improbable that there will be signs of a cavity. Up to this point it will be observed that there is a risk of mistaking the disease either for empyema or for pulmonary tuberculosis, though the bases of the lungs are more frequently affected than the apices. It may be observed that, in order to make confusion more easy, it appears to be quite common for actinomycosis to occur in tubercular people, and sometimes indeed to be actually associated with active tuberculosis of the lungs, and also that it is perhaps impossible at present to diagnose between the pulmonary and pleural cases.

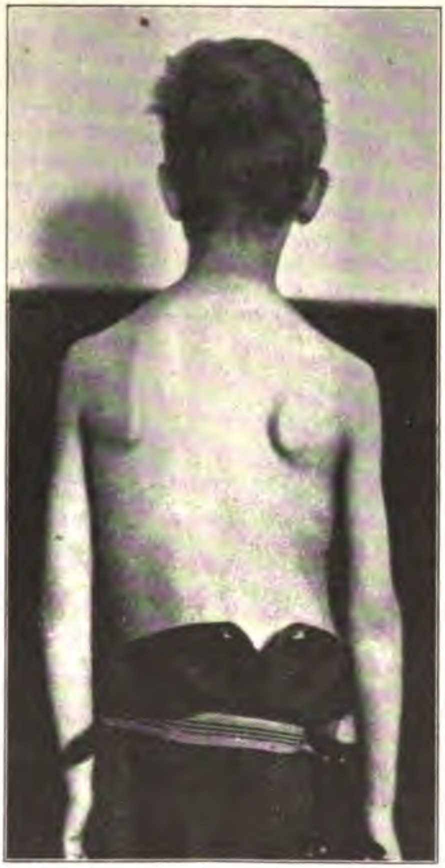


Fig. 115.—Back View of Boy suffering from Actinomycosis of the Right Lung and Pleura in the early stage

Figs. 114 and 115 illustrate the curving of the spine away from the affected side. The later stage of the disease in this patient is shown in Plate 1, fig. 2.

The next stage is reached when the mischief begins to involve the chest walls. An indefinite swelling now occurs, obliterating the intercostal spaces, and gradually involving the superficial and soft structures which become brawny and œdematous; one or more spots in this swelling soften, the skin becomes red, and, if left alone, rupture takes place, with the discharge of a certain amount of pus, probably much less than the amount of the swelling would have led the surgeon to anticipate. The movements of the chest at this period will be decidedly impaired, and the spine will probably be found slightly bent, with the concavity towards the affected side (figs. 114 and 115). At this stage it is obvious that the disease is likely to be mistaken for an empyema or for caries of rib. It is, however, usually easy, after the abscess has burst, to make the diagnosis by the simple process of looking carefully at the pus, or the prominent granulations around the orifice of the sinus either with a magnifying glass, or with the naked eye. Small, pale yellow, roundish granules are seen, sometimes in great abundance, but often not in large numbers. These, though they have been said to bear a resemblance to the crystals of iodoform, can really hardly be mistaken for them, because the colour is paler, and they are obviously not flat, but globular. One of these picked out of the pus or off the granulations with a needle and placed in a little water on a glass slide, and slightly flattened out by pressure with the point of a needle on a cover glass, will generally exhibit, if not in perfection, at all events quite clearly enough for practical purposes, the radiating arrangement of the mycelium, with the club-shaped bodies at the circumference which are characteristic of the disease (figs. 116, 117 and 118). No complicated method of staining is necessary for this purpose, though of course more elegant preparations may be obtained by Gram's and other methods of staining; which, however, do not appear up to the present time to have precisely decided to what class of cryptogamia this organism belongs.

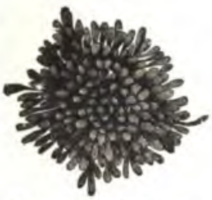


Fig. 116.—Clubs as usually found in Cattle

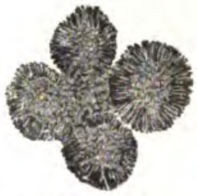


Fig. 117.—Method of spreading (Low power)

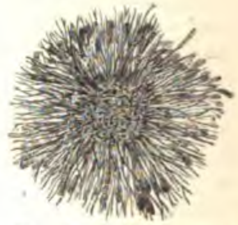


Fig. 118.—Threads as usually found in Man (highly magnified)

In some of the cases described as actinomycosis the mycelium only was found without any of the club-shaped bodies. After studying the next chapter, the reader cannot fail to ask whether it is not possible that some of these would not now have been considered examples of aspergillosis. If the disease has originated in the liver and made its way through the ribs, the external appearances will be the same, but some hepatic enlargement will probably indicate the true origin of the disease (fig. 119).

Course.—The natural tendency of the disease is to advance; and thus, although when it occurs in the mouth a cure is generally if not always obtainable by free removal of the disease, all the early thoracic and abdominal cases ended fatally, because it did not seem possible to attack them surgically, and no treatment by drugs appeared to be of the slightest benefit. Should the disease advance unchecked the patient becomes extremely emaciated, large veins appear upon the surface of the body, and openings are formed in many situations, whilst none of the old ones heal, the temperature remains of the hectic type, appetite is lost, the pulse becomes rapid and feeble, convulsions may occur, and the patient dies a lingering death from exhaustion, probably associated with embolic pyæmia.

DESCRIPTION OF PLATE I.

SHOWING THE APPEARANCE OF THE SORES AND NEIGHBOURING PARTS IN THE TWO CASES OF ACTINOMYCOSIS ILLUSTRATED IN FIGS. 114 AND 119.

Fig. 1, actinomycosis of the liver bursting through the ribs (p. 457, fig. 119). Fig. 2, actinomycosis of the lung and pleura (p. 454, fig. 114). In both *a* indicates the open sore, *b b* the swellings beneath the skin which have not yet given way.

Plate I.

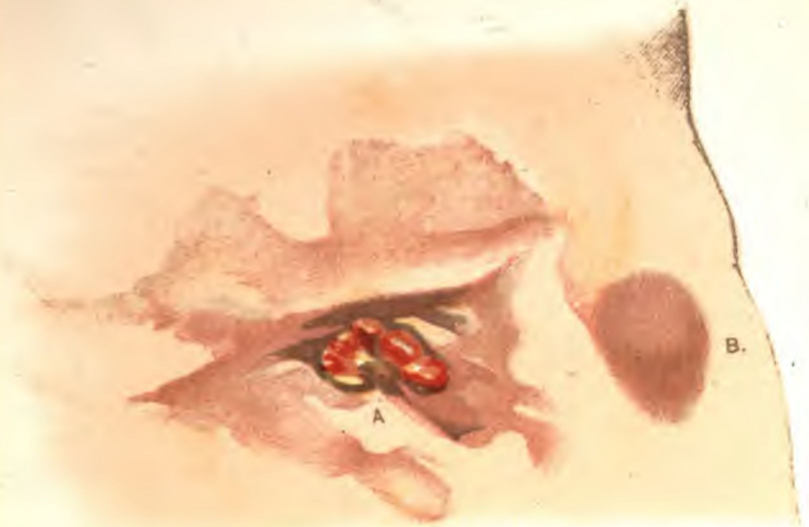


Fig. I.

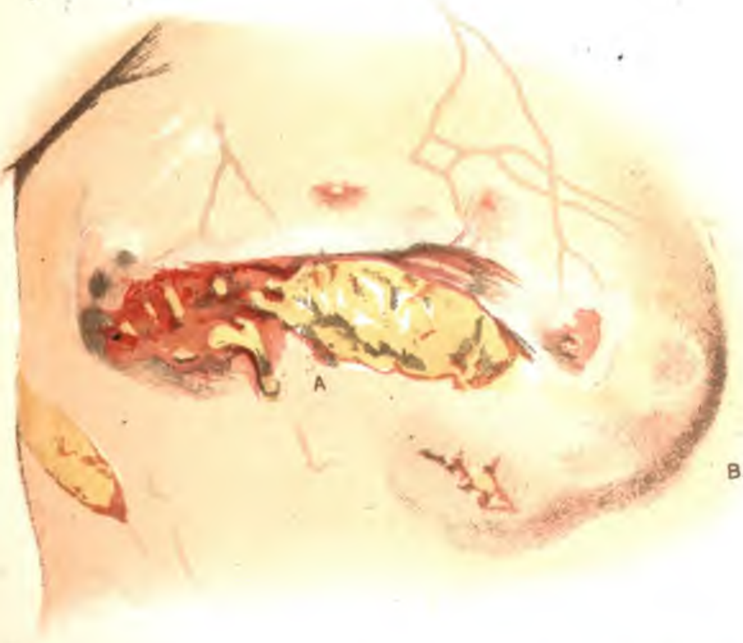
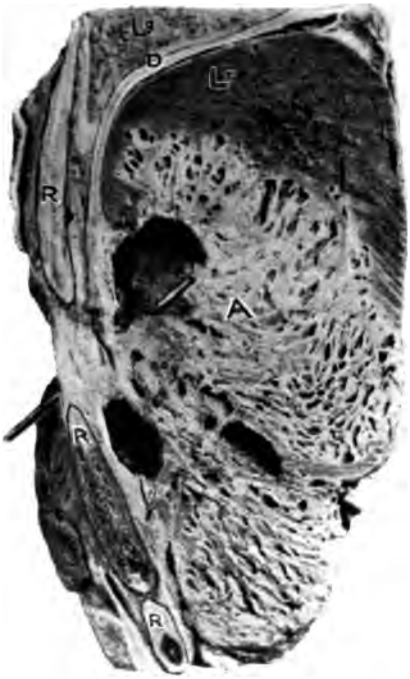


Fig II.

Bale & Danielsson, Lhr



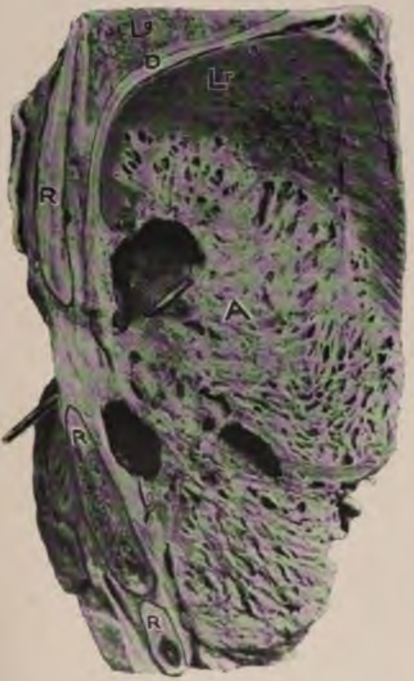
OF THE LIVER OF THE PATIENT SHOWN IN
AND PLATE I., FIG. 1

Lr.	Liver.	A.	Part affected with Actinomycosis
Lg.	Lung.	D.	Diaphragm.

R. R. R. Ribs : the lowest is divided through the **cartilag**

set through the opening in the skin, between the ribs to the ca

PLATE II.



PART OF THE LIVER OF THE PATIENT SHOWN IN FIG. 119 AND PLATE I., FIG. 1

Lr. Liver. A. Part affected with Actinomycosis.
Lg. Lung. D. Diaphragm.
R. R. R. Ribs : the lowest is divided through the cartilage.

The probe passes through the opening in the skin, between the ribs to the cavity in the liver.

The appearance of the wounds and the surrounding tissues in the later stages of the patients shown in figs. 114 and 119 is given in Plate I. The first of these cases is carefully described in the 'Medico-Chirurgical Transactions' for 1889, vol. lxxii. p. 175.

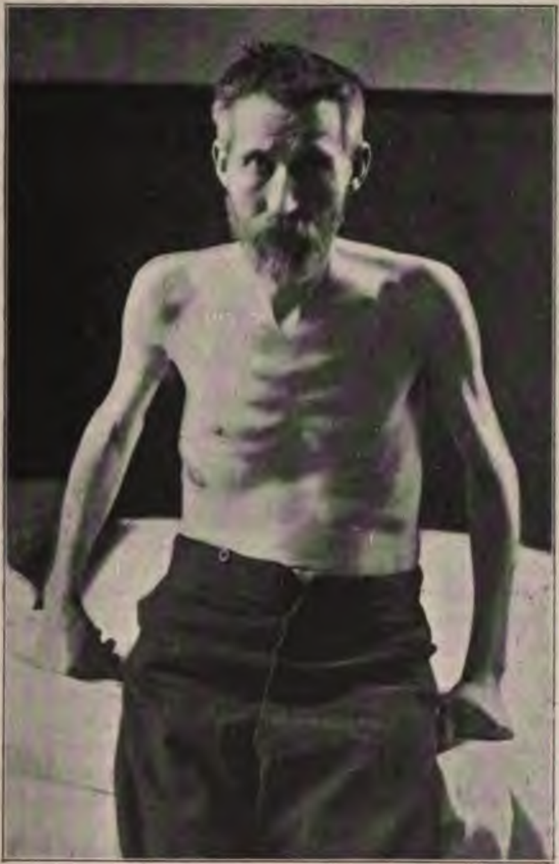


FIG. 119.—FRONT VIEW OF PATIENT SUFFERING FROM ACTINOMYCOSIS OF THE LIVER, AFFECTING SECONDARILY THE RIGHT LUNG AND PLEURA

A prominent mass is seen below and external to the right nipple, in the middle of which there is a sinus which was discharging.

Treatment.—Within recent years the treatment by large doses of iodide of potassium appears to have been of real benefit in a certain number of instances, although in others the disease has continued to advance, if less slowly, towards a fatal termination in spite of the use of the drug. The iodide is well borne and may be given in doses of grs. 30 to 40 three times a day. Still more recently the injection of iodide of potassium has been suggested. We do not think, however, that surgical treatment should be omitted, although the dangers attending it have to be pointed out, and the surgeon must be prepared to find that the growth has extended into regions where it cannot be safely followed; for example, it may run along one or more of the ribs and attack the vertebral column, or may involve large spaces of the pulmonary or hepatic tissue (cf. Pl. I). If an incision be made into it the finger will pass into a soft brain-like material, breaking down with the utmost readiness, and suggesting at first a degenerate malignant growth; but, although this material is yellow and looks as if it were extravascular, it often bleeds with great freedom, and, indeed, hæmorrhage is sometimes so severe as to imperil the life of the patient, who, it must be remembered, is probably, by the time the operation is undertaken, in an exceedingly feeble condition. If, therefore, any operation is commenced, the surgeon should be provided with a rather blunt but fairly large sharp-spoon, and he must have ready a number of plugs, so that, after scraping as much as he deems judicious, he may without any delay arrest the hæmorrhage by pressure. It must be remembered that plugging is not a very satisfactory process when we are dealing with the interior of the lung, not much more satisfactory than it is in the equally common cases where actinomycosis occurs in and about the cæcum; still, notwithstanding the risk of the operation, we are of opinion that, considering the uncertainty of the treatment by iodide of potassium, and the inevitably fatal result if the disease be unchecked, a combination of surgery and medicine is the right course to pursue with this very formidable disease.

What has been said about the hæmorrhage suggests a point which may assist in the diagnosis. If, on opening an abscess which is supposed to be of considerable size, either connected with the rib, or pleural cavity, or spine, or kidney, or cæcum, the amount of pus is found to be unexpectedly small and the hæmorrhage is very free, suspicion should be aroused; and if, after a few days of free drainage, it is found that insidious burrowing of matter is taking place beneath the skin, a careful search should be made for the granules of actinomyces.

R. J. G.

CHAPTER XXXIX

PULMONARY MYCOSIS

(PNEUMOMYCOSIS)

PAGE
Primary aspergillomycosis 460
Aspergillus fumigatus 460
  Pathology and morbid anatomy 460
    Case of Weichselbaum 462
    Case of R. Boyce 463
    Case of Wheaton 464
  Etiology 465
  Symptoms 465
  Physical signs 466
  Diagnosis 466
  Treatment 467
Secondary aspergillomycosis 467
  Etiology 467
  Pathogenesis. 467
  Morbid anatomy 468
  Symptoms, course, and diagnosis. 468
  Treatment 468
Oïdium 468
Mucorinæ 469
Pseudo-tuberculosis hominis streptotricha 469
Appendix: Protozoic infection 469

THE first cases recorded in which the presence of fungi was recognised in the lungs are by Virchow (1854). Since then, from time to time, papers have appeared on the subject, and lately it has received much attention from French writers, particularly Rénon. The diseases of the lungs and bronchi produced by fungi, and particularly the aspergillus fumigatus, have, up to the present date, attracted but little attention in this country, and only a few cases of the kind have been recorded. It is extremely probable that closer investigation of those cases of destructive disease of the lungs which cannot be proved to be due to the action of the bacillus of tubercle will show that some of them are of this nature.

Nodular lesions resembling tubercles are found in the lungs in cases of pneumomycosis, and this fact has led to the use of the term 'pseudo-tuberculosis' as descriptive of such diseases. The term 'tuberculosis' has now a special meaning; it is no longer a disease in which lesions of a certain character are present, *it is a disease due to the action of a specific micro-organism, the bacillus tuberculosis of Koch.* Against this practice we have ventured to protest elsewhere in this work (*vide* p. 379), and have urged that the nomenclature of any newly discovered affections due to the presence of micro-organisms should be based upon their etiology, and not upon the appearances of the lesions produced by the action of the specific virus. We consider that the terms 'tubercle' and 'tuberculosis' should now be regarded as appropriated, and as therefore possessing, when used, a special significance.

The literature of Pneumomycosis is already considerable, but from a practical point of view, which is one we feel bound in this work to consider, the subject is not at present of sufficient importance to justify us in devoting space to its detailed consideration. The reader who is interested in it may be referred to the work of Dr. Rénon,[1] in which the affections produced in animals and mankind by the aspergillus fumigatus are fully and most ably considered. Dr. Rénon's work also contains a very complete bibliography of the subject.

ASPERGILLOMYCOSIS

The *aspergillus fumigatus*, which is the fungus that has been most frequently met with in cases of this kind, is closely allied to *aspergillus glaucus*, the blue mould commonly met with on fruit, cheese, and other decaying matters. Aspergillus glaucus itself is not pathogenic, but some other members of the family appear to be so in dogs and rabbits, and perhaps in other animals. An aspergillus consists of a copious mycelium, like that possessed by most fungi, consisting of delicate, branched, transparent, jointed threads, which ramify in the substance and on the surface of the material on which the fungus is growing. From this mycelium arise other straight threads (carpophores or conidia threads) which bear the fruit. At the summit of these appear rayed divergent protuberances (sterigmata), and attached to each of these is a chain of spores (conidia). At a later period of its history a second kind of fructification is developed from the mycelium, namely a globose receptacle composed of cells, and containing in its interior minute oval bodies called asci, each of which holds eight sporidia.

Aspergillus fumigatus

Pathology and morbid anatomy.—Two views are held as to the nature of this organism: the one, which is supported by Virchow, Spring, and Robin, that it is invariably a saprophyte and that the lesions it produces are secondary to some original disease: the other, which is held by Rénon and Dieulafoy, and which we think the former writer in his work above mentioned has clearly substantiated, is that it may be pathogenic, and the real cause of the lesions found in the lungs and other organs in cases in which it is present, although in other cases it is a secondary lesion.

---

[1] *Etude sur l'aspergillose chez les an* [illegible] *l'homme.* Paris: 1897.

It is possible that in some of the cases which have been described the growth of the fungus may have been a *post-mortem* change. It must also be borne in mind that the aspergillus will grow in some preservative fluids in bottles, e.g. bichromate of potash and Müller's fluid.

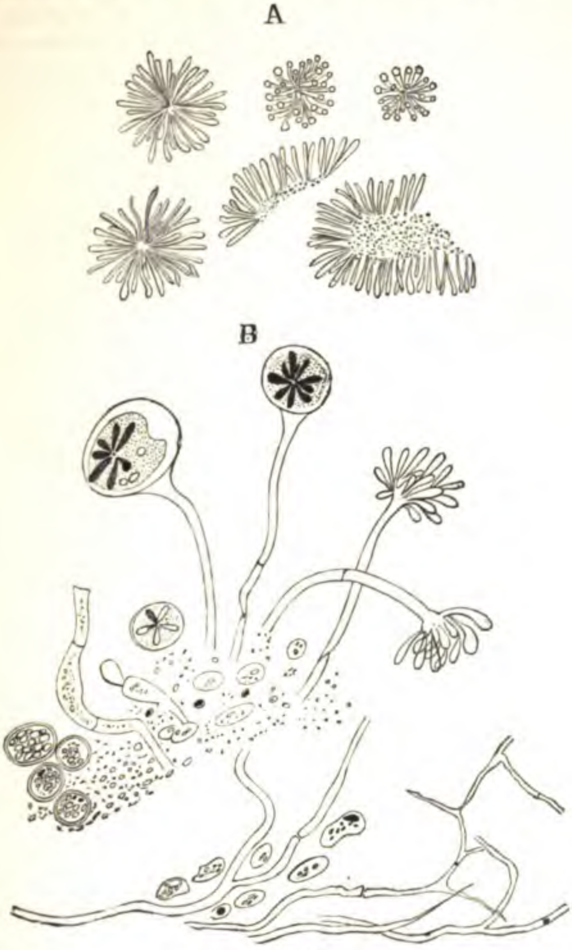


FIG. 120

A, appearances in a scraping from the cut surface of lung, showing rosette-like bodies (× 450) : B, from the patches growing in the right bronchus, showing mycelium and fructification (× 450). Dr. Wheaton's case, 'Path. Soc. Trans.,' vol. xli. p. 34.

with one exception were detached from the basidia, from which they appeared to be formed by segmentation.

Crushed preparations of the harder white bodies projecting from the walls of the cavities showed that they were formed of hyphæ (or threads) thicker than those before described. The walls of the irregular cavities were formed of lung tissue interwoven with hyphæ, which on the inner aspect gave rise to pigmented conidiaphores, and on the outer sent out hyphæ which ramified for a considerable distance in the walls of the alveoli. ('Hyphæ' are the filaments or threads of a fungus which, when matted together, form the mycelium. 'Conidia' are asexual spores, and 'conidiaphores' are the hyphæ which produce conidia. The 'basidia' are large cells borne on the spore-producing surface which give rise to spores at their free ends.)

In the hepatised tissue there were found tubercle-like points and some larger reniform 'pseudo-tubercles' formed of alternating zones of hyphæ of different density. The fibres of the mycelium loosely penetrated the reniform bodies, and spread out in the form of branches into a dense fan-like structure. The whole presented an appearance strikingly resembling that of actinomycosis.

The smaller nodules in some cases started in the alveolar wall, presumably from a spore, as from one thickened central stock numerous irregular processes branched out dichotomously, and formed a mass presenting the appearance of a large mulberry.

The alveoli of the hepatised tissue were filled with an exudation consisting of corpuscles and fibrin.

Near the centre of the lesion the lung tissue and exudation were replaced by necrotic tissue, in which the chromatin granules of broken-down corpuscles were easily recognisable by the deep staining reaction. Leucocytes and macrophages were present, and the latter became more numerous near the aspergillary foci, and especially so near the hyphæ, some of which were completely surrounded by the macrophages. In all the sections a small thrombosed vessel was seen; its wall was infiltrated with hyphæ which had also penetrated the thrombus.

(c) *The form complicated with tuberculosis.*—The case recorded by Wheaton[1] is regarded by Rénon as of this nature, although described by the author as a 'case primarily of tubercle in which a fungus (aspergillus) grew in the bronchi and lung, simulating actinomycosis.'

The patient was a girl, æt. 2½, who had been ailing for two months with a slight cough and marked loss of flesh. There were signs of consolidation of the base of the right lung in front and of the right apex behind.

Eight days after admission signs of breaking down appeared at the base of the right lung posteriorly, and three days later a white patch appeared on the left side of the tongue. Severe continuous pyrexia appeared (103·4°–105·4° F.), and later the temperature reached 106·4°.

---

[1] *Path. Soc. Trans.*, vol. xli. p. 34.

The white patch on the tongue, which had a gelatinous, semitranslucent look, and was very firmly adherent, gradually spread all over the tongue and hard and soft palates. The child died sixteen days after admission.

Post-mortem.—The lower three quarters of the right lung were consolidated in patches which were yellow, mottled with grey. The patches were riddled with small cavities containing pus, and in the surrounding lung were numbers of bright orange-coloured bodies about the size of a mustard seed, which could be picked out with the point of a knife. In the posterior part of the lower lobe there was a cavity the size of a tangerine orange, with irregular black walls covered with white granules. Exposed vessels ran across the cavity, which communicated freely with the main bronchus. In the latter were several white gelatinous patches similar to those on the tongue; they peeled off with difficulty and left raw surfaces. The left lung contained scattered broncho-pneumonic patches and a few orange-coloured granules.

Microscopical examination showed the absence of actinomyces and the appearances were typical of aspergillus.

The mesenteric glands were enlarged, and contained yellow granules similar to those in the lung; in a few places they were suppurating, and the pus contained mycelial filaments. The report states: 'Careful search was made for miliary tubercles, but none were found. . . . On careful search a few tubercles were found in the alveoli of the lung and in the mesenteric glands, but *no bacilli.*' The evidence of the presence of tubercle at all in this case is therefore inconclusive.

Rénon regards the case as certainly one of aspergillus fumigatus, and not aspergillus niger; but he misquotes the title of Dr. Wheaton's paper, as the word 'niger' does not occur there.

A case recorded by Dr. Bristowe[1] may also be consulted.

Etiology.—The disease has been observed up to now chiefly in persons who in some way handle grain, flour, or meal infected with the spores of the aspergillus. Men engaged in the forcible feeding of pigeons in France not uncommonly suffer. Hair-combers who use flour in their work are also liable to be affected. Millers and seedsmen may also possibly furnish examples of this affection. The hair-combers of Paris are stated by Rénon to have noticed that they could not keep certain animals in their houses; birds died from the disease in a fortnight or three weeks, after having suffered from cough and undergone emaciation; dogs lived only three months, but cats proved insusceptible.

Symptoms.—The disease in some cases sets in with hæmoptysis, which may be either slight or profuse, and is generally repeated at intervals. A sense of fatigue and loss of strength are followed by dyspepsia and loss of appetite. Emaciation occurs and cough appears. This is at first dry and recurring in paroxysms; subsequently it is attended by frothy expectoration, which quickly becomes greenish and purulent. The sputum is often blood-streaked.

---

[1] *Path. Soc. Trans.*, vol. v.

Pyrexia is, as a rule, slight, the evening temperature rarely exceeding 38° or 38·5° C. Night sweating is occasionally observed. Dry pleurisy has been present in some cases. These symptoms generally suggest a diagnosis of pulmonary tuberculosis.

Towards the close of the case weakness increases, severe hæmoptysis may occur, and œdema of the legs appears.

In other cases hæmoptysis is slight or absent, the onset being marked by bronchitis, morning vomiting, feebleness and loss of appetite. Dyspnœa then becomes a marked feature and may assume an asthmatic type during the night, when cough is often incessant and accompanied by profuse frothy expectoration.

At a later period the expectoration becomes greenish, purulent, and occasionally nummular.

The **physical signs** in the earlier stages are those of bronchitis, followed at a later period by evidence of consolidation, generally at the apex of one lung.

The *course* of the disease is variable. Periods of improvement are generally observed, during which the patient may gain weight and lose most of the symptoms. Arrest of the disease, accompanied by fibrosis of the lung, frequently occurs, and the aspergillus gradually disappears from the sputum.

Cases have been observed which have lasted three, six, and eight years, or even longer.

The danger most to be feared is the supervention of pulmonary tuberculosis, and the existence of such an affection, and its general similarity to pulmonary tuberculosis, is a proof of the necessity which exists of examining the sputa in all cases of pulmonary disease, a point which we have frequently urged in the course of this work. We have recently heard of a case which, owing to the neglect of this precaution, was overlooked for two months, being regarded as certainly one of tuberculosis.

Diagnosis.—The disease can only be recognised by the discovery of the mycelium or of small rounded white or yellowish-white bodies in the expectoration, although before this its presence may be suspected when the patient is engaged in any of the occupations mentioned above.

The sputa will probably be first examined for tubercle bacilli, under the impression that the case is one of tuberculosis. If none are found, the sputa should be examined for fragments of the mycelium, which may be stained with a watery solution of safranine, or better by staining with thionine (Rénon).

To make certain that the mycelium is that of the aspergillus, fresh sputa should be sown on tubes of sterilised 'liquide de Raulin,' [1] and kept at a temperature of 37° C. If spores or mycelium are present, filaments, at first isolated, but which afterwards unite and form a clump, will be seen to arise on the second day, and slowly to reach the surface of the liquid. Some hours later they form a whitish velvet-like carpet, which twenty hours later is covered with greenish spores which assume a black colour in a few days.

---

[1] For the composition of liquide de Raulin see Rénon, *L'aspergillose*, p. 53

The pathogenic nature of the fungus may be proved by injecting an emulsion of the spores into the axillary vein of a pigeon, or into the vein of the ear of a rabbit. The animal will die in from four to eight days, from generalised nodular lesions in all the viscera, but especially in the kidneys. A fragment of the kidney sown on a tube of 'liquide de Raulin' will reproduce, in from three to six days, a culture of the aspergillus fumigatus.

Treatment.—The indications for treatment are to develop the resisting power of the patient by giving abundance of food, and by enforcing rest during periods of pyrexia. He should be removed to a place where the air is most free from organic impurities. They are, in fact, in all respects similar to those recommended in this work as suitable in the treatment of pulmonary tuberculosis.

There is no specific remedy for the disease known at the present time.

Rénon has found that animals attacked by the disease live longer if iodide of potassium and arsenic are administered to them, and on that ground recommends the use of these drugs in cases in human beings.

The symptomatic treatment of the disease—i.e. of cough, hæmoptysis, and asthmatic paroxysms—should be conducted on the lines elsewhere recommended.

It is possible that the use of creasote vapour baths may prove of service.

In the absence of pyrexia and urgent symptoms, cod-liver oil and tonic remedies should be given.

SECONDARY ASPERGILLOMYCOSIS OF THE LUNGS

Etiology.—The diseases in which secondary infection by the fungus has hitherto been observed are chronic bronchitis, bronchiectasis, broncho-pneumonia, hæmorrhagic infarction of the lungs secondary to valvular disease, pulmonary tuberculosis, and malignant growths of the lung.

In cases of dysentery, cancer of the pylorus, septicæmia, and diabetes, the conditions present appear to favour the growth of the fungus.

Pathogenesis.—Evidence of special exposure to infection by the fungus owing to the nature of the employment, has been lacking in cases in which the disease is secondary to some other affection of the lungs. The cachexia induced by cancer probably acts as a predisposing cause by lowering the resisting power of the patient. In diabetes there may be some special condition present which favours the growth of the fungus. Infection is probably through the air in some cases, in others the organisms were most likely present in the nasal mucous membrane or in the saliva, and have acquired virulent properties owing to some change in the condition of the patient having proved favourable to their growth and multiplication. The fungus has been found in the situations named and in the saliva when no affection of a mycotic nature was present in the individual.

Morbid anatomy.—Rénon describes three forms of lesion: (*a*) bronchial; (*b*) tubercular; (*c*) cavernous.

- (*a*) The growth in this form takes place upon and in the bronchial walls and on the walls of bronchiectatic cavities.
- (*b*) In this variety nodules resembling tubercles are produced in the lung. They are firm, greyish-yellow, well-defined nodules composed of ramified mycelium.
- (*c*) In the cavernous form the growth takes place on the walls of pre-existing cavities in the lung. The fungus is, however, not present in all the cavities—a proof that it is not the cause of the lesions.

The forms resembling actinomyces are never found when the disease is secondary.

Symptoms, Course, and Diagnosis.—The infection usually occurs as a terminal event in secondary cases and hastens the end. The symptoms do not, as in the primary form, undergo remission, as the course of the disease is progressive and a fatal termination appears to be invariable. There are practically no symptoms which can be directly attributed to the presence of the fungus, which is rarely suspected before the discovery of the mycelium in the sputa. This must be sought for and stained according to the methods already described.

Treatment.—The treatment depends upon the nature of the primary disease, and is practically uninfluenced by the presence of the complication.

Oïdium.—Gilchrist and Stokes[1] report a case in which a fungus of this nature was present in the tissues of the skin in a disease resembling lupus, and termed 'pseudo-lupus vulgaris,' affecting the face.

Stained sections of the skin showed hypertrophy of the epidermis, scattered through which were numerous variously sized well-defined miliary abscesses.

In all these abscesses, and also among the granulation cells in the cortex, were numbers of double contoured refractive bodies of a round or ovoid shape. They varied from 10 to 20μ in diameter, many presented buds, and in some a vacuole was observed, but this appearance was not constant; they were usually single, but some were in groups. The contents of the bodies consisted of granular protoplasm; giant cells were present, and the bodies were in some cases contained within these cells. The appearances presented by these bodies closely resemble those described below as found in a case of protozoic infection of the skin. No tubercle bacilli were found.

Pure cultures of the organisms were injected into the external jugular vein of a dog, which was killed in two months.

---

[1] *Bullet. Johns Hopkins Hospital*, vol. vii. No 64: July 1896.

The appearances in the lungs of the dog were as follows :

Projecting from the pleural surface, and completely studding both pulmonary pleuræ, were a large number of firm, light yellow nodules the size of a pea or larger.

They extended from ½ to 2 cm. into the lung. The nodules were round and sharply defined. On section of the lungs both organs were found to be studded throughout with similar nodules. No cavities were present and there were no extensive areas of caseation.

Microscopical examination of the pulmonary nodules showed a central area undergoing coagulative necrosis, in which giant cells were occasionally present. This was surrounded by a zone of large epithelioid and lymphoid cells, the whole forming a nodule to which the term pseudo-tubercle is applied in the report. The rounded bodies in the necrotic areas were stained of a light blue colour. No mycelium was found in the tissues.

Cultivations of the bodies were injected into other animals and a similar disease developed.

The fungus is believed by Mr. Gilchrist to be an oïdium, and allied to the yeast fungus.

Some of the *mucorinæ* or moulds are believed to be pathogenic, but very few cases in which fungi of this class were observed have been at present described.

Pseudo-tuberculosis hominis streptotricha.—Flexner[1] has reported under the above heading a very interesting case in which extensive consolidation, areas of softening, and numerous tubercle-like bodies were present in the lungs. Similar bodies were also scattered all over the peritoneum and in the omentum, which was rolled up and thickened.

No tubercle bacilli were present. Cultures inoculated with material from the lungs showed in twenty-four hours a vigorous growth of a bacillus. The organism is believed to be a new species which Flexner names 'streptothrix pseudo-tuberculosa.'

We mention it in this place owing to the fact that it produces nodules similar to those found in tuberculosis.

J. K. F.

APPENDIX TO CHAPTER XXXIX

PROTOZOIC INFECTION

Rixford and Gilchrist have reported two very interesting cases, which are described as 'Protozoan or Coccidioidal Pseudo-tuberculosis.' For a full account of these cases we must refer the reader to the original report.[2] In the first case, which occurred in a man æt. 40, a farm labourer, the disease began in 1885 as an elevated tender spot on the back of the neck, and increased by the formation of new papillary elevations. The parts chiefly affected were the back of the neck, the eyebrows, nose, cheeks, lips, ears, and hands. The progress of the disease was marked by alternating periods of quiescence and activity.

---

[1] *Johns Hopkins Hospital Bullet.* June 1897.

[2] *Johns Hopkins Hosp. Rep.* 1896, i. 209.

In 1891 a sore, 2½ by 5 inches, was present on the back of the neck, and there was probably also one on the forehead.

The disease remained local until February 1894, when the lymphatic glands of the neck enlarged and developed fluctuation.

Scraping with a sharp spoon was employed in March 1894.

In January 1895 the patient became very somnolent, cough, fever, and emaciation developed, and the physical signs of disease found in the lungs led to the impression that the patient was dying of tuberculosis. The patient died on January 31, 1895.

On post-mortem examination the lungs were anæmic and light-coloured, and to the touch appeared to contain minute masses like grains of sand beneath a membrane. Both lungs were almost completely filled with greyish nodules of nearly uniform size, about one millimetre in diameter, and closely resembling miliary tubercles; combined with these there were caseous nodules and small cavities.

The upper parts of the lungs were more extensively affected than the lower. In the former there were found several small cavities, each containing more or less purulent fluid. Some portions of the lungs were completely consolidated. Many extensive contracting scars were also found in both lungs.

The left apex was adherent to the chest wall, and portions of the pleura were studded with small, greyish, slightly elevated nodules resembling miliary tubercles.

Nodules resembling tubercles were also found on the upper surface of the liver, the peritoneal surface of the diaphragm, the parietal peritoneum of the pelvis and iliac fossa, also in the spleen.

The bronchial glands, the mesenteric glands, the vesiculæ seminales, and the adrenals were enlarged, and, with the exception of the latter, contained pus.

Pus from the testes, prostate, seminal vesicles, a sinus in the tibia, and an abscess in the hand contained numbers of protozoa similar to those which during life had been found in the lesions of the skin.

The fluid in the pulmonary cavities contained immense numbers of protozoa, and similar bodies were present in abundance in the lung tissue, and also in the suprarenal capsules and the lymphatic glands.

Microscopically, the tubercular-looking nodules were found to present a histological structure precisely similar to that of tubercles, the resemblance being so close as practically to amount to histological identity.

The tubercle-like bodies not only presented the same varieties of cells arranged in the same manner as are found in true tubercles—viz. epithelioid cells, giant cells, and lymphoid cells—but they were the seat of coagulative necrosis or caseation. By the kindness of Mr. Gilchrist we are able to give an illustration showing the appearances presented by these bodies.

The organisms were present, often in considerable numbers, in the giant cells, and were scattered through the lung. Sporulating forms were fairly numerous.

The appearances presented by the diseased lungs were practically similar to those found in tuberculosis, except that the tubercle bacilli were absent and their place was taken by the protozoa.

The adult encapsulated organisms showed a distinct double-contoured capsule; they were almost spherical, and varied from 15μ–27μ in diameter. The capsule was thick and well defined, and stained deeply. Between the capsule and the contents there was a clear refractive layer, which either stained with difficulty or more often did not stain at all.

This clear zone appears to be homogeneous and structureless; it varies in thickness from 1μ–3μ, and is hardly discoverable when sporulation is in process.

The protoplasm of the organism is surrounded by the clear layer, and stains readily; it contains fine and some scattered coarse granules; the latter are sometimes seen at the periphery, or they may form a network.

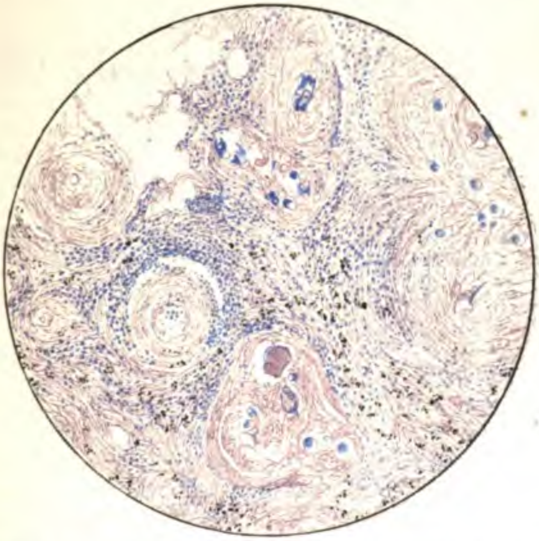


FIG. 123.—SECTION OF LUNG FROM A CASE OF PROTOZOIC INFECTION SECONDARY TO DISEASE OF THE SKIN

The proof afforded by these observations that lesions, which to the naked eye and upon microscopical examination cannot be distinguished from those resulting from the presence of the bacillus tuberculosis of Koch, may be produced in the lungs by infection from fungi and protozoa is complete, and shows how absolutely necessary it is in all cases of suspected tuberculosis to search for the typical bacillus, and not to draw conclusions as to the nature of the disease merely from naked-eye or histological evidence.

J. K. F.

CHAPTER XL

HYDATID DISEASE OF THE LUNGS

	PAGE
Etiology	473
Morbid anatomy	473
Characters of hydatid fluid	474
Subsequent changes	474
Symptoms and physical signs	475
Before rupture	475
After rupture	476
Diagnosis	477
Prognosis	478
Treatment	479

HYDATID disease of the lungs, like that of other organs, is due to the introduction into the body of the ovum of the tænia echinococcus.

Out of 809 cases of single hydatid cyst occurring in the Australasian hospitals collected by Thomas[1] the thoracic organs were affected in 141 cases as follows:

Lung	134 cases
Pleura	2 „
Heart	4 „
'Thorax'	1 case
	141

Hydatids appear to be met with in the lungs in a larger proportion of cases in Australasia than in Europe, as is seen by a comparison of the tables of Neisser and Thomas.[2]

Organ	Proportion per cent. of total cases		Ratio of lung to liver cases
	Neisser's table: Europe	Thomas's tables: Australasia	
Liver	50·11	65·76	In Europe, 1 to 6½
Lung	7·44	16·56	In Australasia, 1 to 4

---

[1] *Hydatid Disease.* Sydney: 1894.

[2] *Op. cit.*, p. 122.

Etiology.—The disease is most common in countries, such as Australia and Iceland, in which infected dogs are numerous, and associate closely with human beings in their domestic life. Water containing the ova of the worm is probably the chief source of infection.

Morbid anatomy.—A single hydatid cyst is generally present, but both lungs may be affected, and there may be more than one cyst in a single lung. Cysts in other organs are frequently met with. The right lung is more often affected than the left (right, 121 cases; left, 74 cases: Thomas), and the lower lobes of the lungs than the upper. The multilocular variety of hydatid has been met with in the lungs, but it is of very rare occurrence.

The ovum of the tænia echinococcus having been taken into the stomach, its chitinous envelope is dissolved by the aid of warmth and the action of the gastric juice, and the liberated ovum bores through the coats of the stomach or intestine, and probably in the majority of cases of hydatid disease enters a branch of the portal vein and is carried in the blood stream to the liver.

In cases in which it becomes arrested in the lungs it is possible that it may have already passed through the capillaries of the portal and hepatic veins, or it may have entered a systemic vein after leaving the stomach. In either case it reaches the lungs through the pulmonary artery.

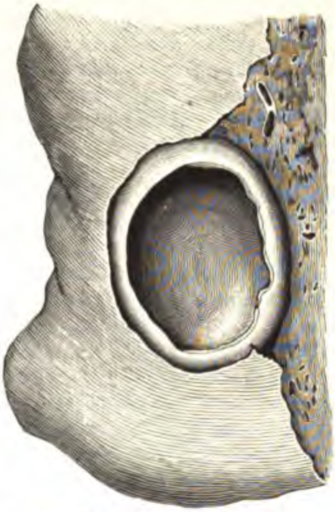


FIG. 124.—HYDATID CYST IN THE LUNG

Dr. Bird[1] suggests that the greater prevalence of hydatid disease of the lungs in Australia than in Europe is due to the inhalation of dust containing the ova of the echinococcus. If this is the mode of infection the ova may possibly reach the lungs through the bronchial arteries; but the share taken by the processes of vascular transportation and migration respectively in the distribution of the ova of the tænia echinococcus must be considered doubtful.

The changes which the ovum undergoes in the lungs are similar to those which occur in other organs. It loses its external hooks and is gradually converted into a cyst containing fluid. The cyst wall is composed of a thick transparent elastic laminated cuticle (ectocyst) and an internal granular or parenchymatous layer (endocyst).

---

[1] *On Hydatids of the Lungs.* Melbourne: 1877.

Characters of hydatid fluid.—The fluid is clear, transparent, colourless, and neutral; the specific gravity varies between 1·005 and 1·015; chloride of sodium is present in considerable amount, and there may be traces of sugar; but, so long as the cyst is living, there is either no albumen or only an infinitesimally small quantity.

In the lungs, as in other organs, brood capsules, scolices, and ultimately daughter cysts may be formed by a process of budding from the granular or parenchymatous layer of the cyst wall, or the cyst may be sterile.

The presence of the cyst usually excites some inflammatory change in the surrounding tissue, and thus an adventitious capsule is formed; but, except in cases of prolonged duration, this is rarely of the dense fibrous character commonly found in hydatids of the liver. There may indeed be no capsule at all, the lung tissue around being either normal, or only slightly condensed as a result of pressure. The frequent absence of a definite lining membrane to the cavity occupied by a hydatid cyst in the lungs is a fact of much importance.

As the cyst enlarges it displaces the lung and erodes the tissue around, so that small or large bronchi may open directly on to the surface of the cyst; but, so long as it is kept tense by the pressure of the fluid within it, these apertures are closed. If, however, the cyst becomes collapsed, either from rupture or puncture, as in the operation of paracentesis, the contents of the cyst are discharged into the bronchi and air enters the cavity.

Subsequent changes.—*Rupture into a bronchus* is the most common event. It is followed by the discharge of the contents of the cyst, and possibly also of the cyst itself, in which case the cavity in which it was lying may be gradually obliterated and the disease thus undergo a cure. This favourable result is, however, of rare occurrence, and is never observed when the adventitious cyst wall is firm and fibroid, as under these circumstances the cavity remains and continues to discharge pus.

Rupture into the pleura is usually attended with pneumothorax. The subsequent occurrence of inflammation of the pleura gives rise either to a serous or a purulent effusion, the appearances of which will be to some extent modified by the condition of the fluid contents of the sac previous to its rupture.

Rupture into the pericardium may be quickly followed by death.

Pneumonia may occur in the tissue around, and may pass into gangrene; but such an event is rare so long as the cyst is living. The pneumonic process may become chronic and be followed by fibroid induration of the consolidated part of the lung.

Suppuration.—Inflammatory changes in the adventitious capsule may lead to the death of the hydatid, and this may be followed by suppuration of the cyst and fœtid decomposition of the sac and its contents. The condition is then one of pulmonary abscess, and rupture into a bronchus or into the pleura is very likely to occur. The cyst may, however, burrow through the diaphragm and form a sac between it and the liver. This may be followed by the discharge of the contents of the cyst through an opening at the umbilicus.

After the death of the hydatid the fluid becomes turbid and albuminous. The mother cyst first presents a glutinous appearance, then becomes opaque, and ultimately breaks up into fragments. Daughter cysts, if present, undergo similar changes, but at a rather later period than the parent cyst.

Spontaneous retrogression of a hydatid cyst is not uncommonly observed in the liver, but is an event of rare occurrence in the lungs, especially after the cyst has attained a large size. In such cases the contents of the cyst undergo fatty degeneration, a putty-like mass is formed, and the cyst shrivels.

Symptoms before rupture.—The cyst may give rise to few or no symptoms so long as it is small and centrally situated; but, as it increases in size, *cough* is usually induced. This is generally dry and hacking, but may be accompanied by some mucoid expectoration. The cough is occasionally paroxysmal in character, and may resemble that of whooping cough.

Hæmoptysis takes place in the majority of cases; it is usually small in quantity during this stage, the blood merely staining the expectoration; but profuse hæmoptysis may occur before rupture of the cyst. Owing to the slow growth of the cyst the lung accommodates itself to its presence and *dyspnœa* is rarely severe; it may still be slight in degree even when the cyst has attained a large size. Urgent dyspnœa is often the result of pressure upon a large vessel.

Pain is rarely severe, and may be absent, or there may be a sense of weight in the chest. The onset of pain is sometimes an indication that the cyst has reached the surface of the lung and has involved the pleura.

So long as no inflammatory changes are excited in the lung or pleura, there is no *pyrexia*. The general health of the patient may be but slightly affected, although some degree of loss of flesh generally occurs.

The physical signs before rupture of the cyst depend upon its size and position in the lung. If the cyst is small and deeply seated, nothing abnormal may be found. If it is of considerable size and near the surface of the lung, but separated from the pleura by compressed lung, the percussion note over it may be of a subtympanitic or skodaic character; the expansion of the affected side is diminished, the breath sounds weak, and the cardiac impulse may be displaced. If the cyst is in contact with the pleura the chest wall at that spot may be rounded and the intercostal depressions obliterated. The vocal fremitus will be feeble or absent. The most characteristic physical sign is a rounded area of dulness on percussion, the note obtained being absolutely dull in the centre of the area and gradually increasing in resonance toward its margin, the breath sounds and vocal resonance over the same site being absent.

The cyst, after coming into contact with the pleura, may in the course of its further enlargement project through an intersp and form a globular *tumour on the surface of the chest*—a very important and characteristic sign of the disease. Over such a tumour, or where no swelling of the kind is present, a thrill may be felt on percussion—the so-called hydatid thrill—a sign which is, however, not limited to fluid-containing cavities of this nature.

Some crepitant râles may be caused by congestion or œdema of lung tissue in the neighbourhood, and the *cardiac impulse may be displaced*. Displacement of the heart may, in some cases, be almost the only abnormal sign for a long period, as occurred, for example, in a case lately under observation at the Brompton Hospital, in which the heart had been markedly displaced for six years previous to the onset of an attack of double pneumonia, during which the real nature of the disease was discovered.

Signs of pressure upon the vessels, pneumogastric nerves, or œsophagus are very rarely met with. A cyrtometric tracing may show enlargement of the affected side when there is no definite tumour.

It may prove to be possible, by the aid of the Röntgen rays, to determine the presence and exact position of a large hydatid cyst in the lungs.

The *rupture of the cyst into a bronchus* is accompanied by urgent symptoms which may have an immediately fatal termination. Rupture is usually attended by violent cough, severe pain in the chest, urgent dyspnœa, and the expectoration of a quantity of watery fluid containing hooklets, and possibly by severe hæmoptysis. If daughter cysts and pieces of hydatid membrane are found in the expectoration it shows that the bronchus in communication with the hydatid cavity is a large one. If the opening into the bronchus is small the escape of the contents of the cyst may be more gradual and the symptoms immediately attending its rupture less severe. Copious hæmorrhage from the vessels of the adventitious capsule may occur not only at the time of rupture, but on frequent occasions during the course of the subsequent illness.

Symptoms after rupture.—If the patient recovers from these symptoms cough continues and is usually violent, a severe exacerbation attending each fresh escape of daughter cysts and fragments of membrane into the bronchi. The expectoration often becomes fœtid and purulent from suppuration within the cavity; in chronic cases it may be chocolate coloured and may resemble very closely the pus which is supposed to be characteristic of an hepatic abscess. Severe pyrexia of a hectic type usually accompanies suppuration of the cyst, and under such circumstances there may be considerable emaciation, and clubbing of the fingers may occur.

In some cases, however, the symptoms which follow rupture of the cyst into a bronchus are much less severe; and although quantities of cysts and membrane are expectorated during many months, if fever is absent, the general health may not be seriously impaired. The expectoration of the cysts may, however, be attended by most urgent attacks of dyspnœa owing to their temporary impaction in

the glottis, and at such times there may be danger of the occurrence of asphyxia.

Physical signs.—After rupture. The signs of a cavity may usually be discovered, but in some cases it is impossible, owing to the contraction of the cavity which contained the cyst, to determine its site by physical examination of the chest. The attempt is most likely to be successful if the chest is auscultated during or immediately after an attack of coughing, accompanied by expectoration.

Rupture into the pleura is attended by sudden severe pain, urgent dyspnœa, and possibly by collapse. Physical signs indicating the presence of fluid, and probably also of air, will immediately be present, and the heart will be markedly displaced to the opposite side.

Rupture into the pericardium is attended by severe pain in the chest and evidence of great embarrassment of the action of the heart, symptoms which may be quickly followed by death. The chief physical signs will be marked increase in the area of precordial dulness and absence of the cardiac impulse.

Diagnosis.—Before rupture. The nature of the case may be suspected if the percussion signs already described as typical of the disease are present; but until they appear or rupture takes place a diagnosis may be impossible.

In a case under the care of the late Dr. Bristowe, a correct diagnosis was made from the fact that, although all the physical signs pointed to the presence of a pleural effusion, *a friction sound was audible all over the area of dulness.*

Displacement of the heart, for which no cause can be discovered, should suggest the possible presence of hydatids. In some cases the discovery of hydatids in other organs has suggested the true cause of obscure pulmonary symptoms. The diagnosis from tubercular disease of the lungs is to be made by close attention to the site of the lesions, the fact that they have not followed the 'line of march,' and the absence of tubercle bacilli from the sputa.

The general health is, as a rule, far more seriously affected in pulmonary tuberculosis than when an unruptured hydatid cyst is present in the lung.

After rupture.—The presence of hydatid disease will be suspected from the history of the symptoms attending the rupture of the cyst, and this suspicion may be confirmed by the discovery in the sputa of hooklets or fragments of hydatid membrane.

If an [illegible] hooklets [illegible] be difficult [illegible] monary [illegible] shows [illegible] occur after [illegible] met with.

If the [illegible] attempt

should be made to convert suspicion into proof by the puncture of the chest with an aspirator needle, at any rate until the patient is on the operating table and under the influence of an anæsthetic, and every preparation has been made for an immediate incision should it prove to be necessary, as death has been known to follow from suffocation caused by the escape of the fluid into the bronchi.

When a hydatid cyst is situated in the upper part of the right lobe of the liver and projects towards the thorax, there may be great difficulty in arriving at a diagnosis, as a cyst in the lower lobe of the right lung or an effusion at the base of the right pleura may give rise to very similar physical signs. If jaundice is present and the liver projects considerably below the costal margin, the cyst is probably situated in the liver; but jaundice is often absent in hydatid disease of the liver, and the liver may be enlarged from some independent cause when a cyst is situated in the lung. If the upper line of the dulness is highest in the axilla and slopes downwards both towards the sternum and towards the spine, the probability is in favour of the presence of a pleural effusion, whereas if the upper level of the dulness at the right base is rounded there may be a cyst in the liver projecting upwards, or the cyst may be situated in the lower lobe of the right lung; but it may be possible by the aid of this sign to exclude the presence of a pleural effusion.

If a hydatid cyst of the liver perforates the lung and the contents of the cyst are expectorated, a diagnosis of the primary seat of the hydatid may usually be made from the fact that the sputa are bile stained.

Hydatid cysts of the kidney have also been known to perforate the lung. The diagnosis of such a condition may be made by close attention to the history of the case, and particularly by the fact that the tumour was primarily seated in the lumbar region.

The absence of pressure upon the large vessels, trachea, main bronchi, pneumogastric nerves, and œsophagus are signs of most importance in the differential diagnosis of hydatid of the lung from other forms of intrathoracic tumour. If a pleural effusion can be excluded, and fluctuation is present, the case is almost certainly one of hydatid disease, as that sign is not found in any other affection which is limited to the lung and does not involve the pleura.

Prognosis.—If a correct diagnosis is made before rupture of the sac occurs, the prospects of recovery after operation are good, as a very large proportion of cases treated by incision and drainage are cured.

The chances of spontaneous cure taking place without rupture are so remote as to be excluded from consideration when there is good reason to suspect the presence of a hydatid in the lung.

If death does not occur at the time of rupture of the cyst into a bronchus or elsewhere, the prospect of ultimate recovery is fairly good, as it follows in rather more than half the number of cases. The prognosis in such cases is chiefly determined by the character of the general symptoms, particularly the degree of fever, and the extent of emaciation. Frequently repeated hæmoptysis is an unfavourable condition.

If the discharge of the contents of the cyst has been preceded by the death of the hydatid and a fœtid abscess has resulted, the prognosis is much less favourable. It will depend chiefly upon the possibility of discovering the site of the cavity and of draining it effectually.

Treatment.—Medical treatment is quite useless except to alleviate symptoms.

Paracentesis, except as a preliminary step at the time of operation, is absolutely inadmissible. It has no doubt, in a certain number of cases, led to a cure without further operation, but the risk is too great to warrant such a procedure.

For the sake of convenience the surgical treatment of hydatids is reserved for the chapter on Hydatids of the Pleura.

J. K. F.

CHAPTER XLI

HYDATIDS OF THE PLEURA

PAGE
Morbid anatomy 480
Symptoms 480
Physical signs 481
Diagnosis 481
Course and prognosis 481
Treatment 482

THE pleura is rarely the primary seat of hydatids. In some cases which are apparently of this nature it is probable that the cyst has been formed within the lung, but near its surface, and has thus involved the pleura at an earlier period than usual.

Morbid anatomy.—A hydatid of the pleura is usually single; it varies in size with the duration of the disease; if long unrecognised, the cyst may become so large as to cause almost complete collapse of the lung and may contain three or four pints of fluid. As much as nine pints of fluid have been removed from a cyst which was believed to be of pleural origin.

Hydatids of the pleura, as a rule, merely compress the lung, but perforation of the lung may occur and be followed by the discharge of the contents of the cyst through the bronchi.

Symptoms.—Pain is a more prominent symptom, and occurs at an earlier period, when a hydatid is situated in the pleura, than when it is in the lung; it is often constant, and may be severe.

Cough may be absent and hæmoptysis rarely occurs.

The degree of dyspnœa depends upon the size of the cyst; it is seldom severe, even when a large cyst has produced collapse of a considerable area of the lungs.

There may be no fever, provided that the contents of the cyst are aseptic, and inflammation of the pleura is not excited. The occurrence of suppuration in the hydatid is accompanied by severe pyrexia of a hectic type, and, in some cases, by rigors.

A hydatid may occupy the pleura for a long time without producing any marked effect upon the general health, but emaciation and sweating usually occur when the cyst has attained a large size.

Physical signs.—The description of the physical signs of hydatid of the lungs may be consulted, as what is there stated applies with slight modification to hydatid disease of the pleura. There may, as in hydatid of the lung, be a localised globular swelling or a general bulging of the side with obliteration or prominence of the interspaces. Expansion of the affected side is either diminished or absent.

The percussion note over the area occupied by the cyst is more completely dull, and fluctuation is more readily obtained than when the cyst is situated within the lung. When a large cyst occupies the lower part of the pleura, the dulness may extend to the fourth or third rib in front and to the angle of the scapula behind, or a dull note may be obtained over a still larger area of the chest wall. A cyst in this situation may also cause a bulging in the epigastrium.

Localised inflammation of the pleura is apt to occur from time to time during the course of the disease, and at such periods a friction sound may be present. The breath sounds and vocal fremitus and resonance will be absent over the dull area, and the cardiac apex will be displaced.

Diagnosis.—A hydatid of the pleura is often mistaken for a pleural effusion, a correct diagnosis being rarely made until some of the fluid has been withdrawn from the cyst or perforation of the lung has occurred.

If the contents of the hydatid are aseptic, there will probably be no fever, and if so that fact will help to differentiate the case from one of inflammatory effusion into the pleura; but if suppuration has occurred in the cyst and there is pyrexia of a hectic type the similarity of the condition to empyema will be very close.

The upper line of the dulness may be rounded in a hydatid, whereas in most cases of pleural effusion it is highest in the axilla and slopes downwards towards the anterior and posterior aspects of the chest, but a localised empyema may present a rounded upper line of dulness.

The presence of a hydatid of the pleura may be suspected when, although the heart is markedly displaced, the area of dulness is limited in extent, and suggests that the pleura contains only a moderate quantity of fluid.

Course and prognosis.—If the nature of the disease is recognised at an early period and proper treatment is adopted, the prospects of complete recovery following the evacuation of the contents of the cyst and the removal of the cyst wall are very favourable.

The lung, although extensively collapsed from compression, may re-expand immediately the cyst is emptied. This has been known to occur at the time of operation during the act of coughing. If suppuration has taken place in the cyst, the prospects of complete recovery after operation are less favourable, as a discharging sinus may remain, and amyloid disease of the viscera may be induced, but such a mode of termination of the disease is rare. If perforation of the lung occurs, the prognosis is rendered much less favourable, while rupture into the pericardium may be immediately followed by death.

J. K. F.

SURGICAL TREATMENT OF HYDATIDS OF THE LUNG OR PLEURA

The presence of hydatid, either of the pleura or of the lung, having been suspected, the treatment will vary according to the condition of the parasite. If it be merely suspected from the physical signs that it is a living cyst distended with clear fluid, unaccompanied by daughter cysts, the question arises as to the safety of puncturing it with a needle attached to an aspirator, with the view of confirming the diagnosis, and possibly of treating the disease. It is, of course, an exceedingly tempting thing to do, the employment of the aspirator being so simple, and, in the majority of cases, its use being totally devoid of danger. It becomes still more tempting when we read the published accounts by Australian surgeons of the large number of cases in which a simple puncture of hydatid in the lung has been followed by a complete cure, such as has often, but not invariably, followed similar treatment applied to other parts of the body. We hear, however, together with these accounts of brilliant success, a note of warning in respect of a very grave danger with which puncture is associated, viz., that, owing to the sudden escape of fluid into the cavity in which the cyst is lying, and thence into the bronchi opening into it, the patient has been, so to speak, drowned by the rush of fluid into the air passages. Should the surgeon, therefore, be tempted to employ the aspirator, the use of which is probably attended with greater danger than that of a simple trocar and cannula, he should never do so without having all his instruments ready for immediate incision in case this accident should occur; and should it do so, he must without hesitation plunge his knife freely into the cyst and at once adopt the recognised remedies for the resuscitation of the drowned. It will be seen, therefore, that it is open to question whether it is not always wiser to proceed methodically to the opening of intrathoracic cysts when there are reasonable grounds to suspect their presence—a conclusion which for other though somewhat similar reasons, many surgeons have come to in regard to the treatment of hydatids in the abdomen. Of course, it cannot be denied that the diagnosis of the latter is much more easy than that of the former.

The steps of the operation will not differ from those employed in opening an abscess in the lung (see p. 419). Should the hydatid be living and situated in the pleura, the proceeding will be extremely simple; should it, however, be living and embedded in the substance of the lung, a preliminary fixing of the lung to the pleura (see p. 419) must be undertaken. I have not at present had to deal with any case of this description.

No difficulty with regard to the best line of treatment to be followed exists in that other class of cases, where the hydatid is already dead and the diagnosis is no longer doubtful because the patient has expectorated, or is expectorating, fluid containing the recognisable elements of the parasite. It may possibly be difficult to localise the position of the cyst, but if, after a considerable time, the expectoration continues, an attempt should be made to effect a cure by means of an external opening. If there is marked dulness in the usual situation, namely, at the posterior base of the right lung, it is probable that no more difficulties will be met with than in the opening of a simple empyema; but it has happened to me, after evacuating a cavity, which was obviously formed from part of the pleural cavity, to find beneath it an old dense-walled hydatid cyst, which was evidently in the substance of the lung. Should, however, the dulness be very slight, and the localising symptoms only consist of a patch of somewhat diminished resonance, over which moist râles are to be heard, it is more than doubtful whether adhesions will have formed between the lung and the chest wall, and under these circumstances the cautious method of treatment already so often referred to (see page 419) should be followed in all its details; for if the surgeon omit to stitch the lung to the chest wall, he may possibly lose his chance of reaching the hydatid altogether.

It must not be forgotten that hydatids may be met with at any part of either lung. A case is referred to on p. 642 in which one was found on the under surface of the base of the right lung, which had produced a hydro-pneumothorax, and in which it was possible to remove the whole cyst. The question has been discussed as to whether an attempt should be made to remove the cyst as a routine practice, or whether we should be content with draining the cavity. The opinions of pathologists differ as to the degree of fibrous capsule which is likely to be met with. I feel sure that in old cases nothing is to be gained by attempting the removal of the firmly attached ectocyst, which, in my small experience, has always been present, and that much danger may be incurred by doing so. In recent cases, as far as I have seen, there is very little fibrous capsule, and the endocyst comes away quite readily from the surrounding scarcely indurated lung tissue.

It is important to remember that it is very common for more than one hydatid to be present in the same patient, and also that hydatids of the liver not very unfrequently make their way into the lung or into the pleura. In the latter case it is likely that the diagnosis will be helped by the presence of bile in the fluid which is expectorated or evacuated, but it must not be forgotten that the expectoration from an old hydatid of the lung may be so very like the matter derived from an hepatic abscess as to suggest a mistaken diagnosis. Of this I have had painful personal experience. A middle-aged man had bloodstained purulent expectoration containing booklets; there was a very indefinite patch of diminished resonance, with a very few moist râles at the base of the right lung behind; there was also a rounded swelling on the front of the left lobe of the liver. It was thought likely that the expectoration came from the hepatic swelling, but it turned out that this was a living hydatid cyst, and that the pus came from an old dead cyst in the right lung.

Experience of European surgeons on this subject is still so meagre that we have in the text books very dismal accounts of the prognosis in such cases. My own experience does not extend to more than five or six cases, but so far the results have been satisfactory, and the experience of our Australian colleagues should encourage us to avoid too dismal a prognosis.

R. J. G.

CHAPTER XLII

TUMOURS OF THE LUNGS

PAGE
Sarcoma and cancer 485
  Site of the disease 486
  Morbid anatomy 487
  Etiology 487
  Symptoms 488
  Physical signs 488
  Diagnosis 489
  Duration and prognosis 489
  Treatment 489
Removal of portions of the lung or pleura for tumours and other morbid conditions 489

SARCOMA AND CANCER

PRIMARY malignant disease of the lung is a rare condition; but secondary deposits of sarcoma and cancer are of more frequent occurrence. An analysis of 112 cases of tumours of the lung recorded in the 'Transactions of the Pathological Society,' made by Wilson Fox, showed that the disease was primary in 38 and secondary in 64. The remaining 10 could not be thus classified. Of the former, 14 are classed as encephaloid, and 12 as 'cancer.' In three cases the growth was an enchondroma; in five, lymphadenoma; in two a sarcoma; and one is described as 'fungus hæmatodes.'

These cases are spread over a period which has been marked by great changes of view as to the exact nature of new growths, and if they could be re-examined at the present time it is very probable that many which are classed as 'cancers' would now be regarded as sarcomata. The mention of such a large number of cases of primary tumour of the lung may convey the impression that such growths are more common than is really the case; as a fact they are extremely rare, the large majority of new growths within the thorax being of mediastinal origin.

The Middlesex Hospital Report for 1888 contains an analysis by Mr. W. Roger Williams of 890 cases of cancer, of which 317 were examined post-mortem.

The following table shows the frequency with which secondary deposits were present in the lungs:

Site of disease	Number of autopsies	Lungs			Total
		Right	Left	Both	
Uterus	83	1	0	6	7
Breast	45	1	2	5	8
Tongue and mouth	61	0	0	5	5
Lip	11	1	1	—	2
Rectum	29	1	1	5	7
Œsophagus	16	1	—	—	1
Pharynx	2	1	—	—	1
External genitals	11	—	—	2	2
Ovaries	9	2	—	—	2
Stomach	3	—	—	—	0
Peritoneum	4	—	—	—	0
Colon	8	—	—	1	1
Skin	6	0	1	2	3

The small number of cases of cancer of certain organs in which malignant disease is common, *e.g.* the stomach, is explained by the fact that only the cases from the surgical and cancer wards are included in this table.

In 29 autopsies upon cases of cancer of the anus, testes, bladder, prostate, ileo-cæcal valve and cæcum, superior maxilla, submaxillary region, larynx, and in four cases of rodent ulcer no secondary deposits were found; but in some of these cases, and especially when the primary disease involved the œsophagus, the intra-thoracic organs were involved by direct extension.

Primary cancerous growths may originate in the mucous glands of the bronchi and may thus acquire the characters of a cylindrical epithelioma. The alveolar epithelium is another possible site of origin. In such cases the growth presents a typical alveolar arrangement with cells of a spheroidal shape, and it may contain inhaled pigment. The growths appear to spread in this as in other organs along the lymph channels.

Sarcomata may also be primary, but are more often secondary, and not uncommonly appear some time after the removal of the primary tumour.

The round-celled variety is most frequently met with, but spindle-celled and myeloid growths also occur and secondary deposits of melanotic and osteo-sarcoma are also not uncommonly observed in the lungs.

Secondary growths, whether of cancer or sarcoma, reproduce the type of the disease from which they originated.

Enchondroma.—Growths of this character are of such rare occurrence in the lungs that we must be content with the mention of the fact that they are occasionally met with, and must refer the reader for a full consideration of the subject to works on pathological anatomy. Enchondroma may originate in the cartilages of the bronchi.

Morbid anatomy.—*Primary* growths may appear either as definite tumours or as infiltrations, the latter being the more common form. A large mass of new growth may occupy an extensive area of the lung and almost destroy all traces of the pulmonary tissue, or a growth may extend into the lung along the peribronchial and perivascular lymphatic spaces, and in the interlobular septa.

In some cases both forms of growth are combined.

A *cancerous growth* may form a hard solid white mass of almost uniform aspect, or it may be soft and fleshy-looking. Areas of pigmentation, which are often present in infiltrating growths, mark the site of bronchial glands.

Sarcomata may appear as two or three masses of considerable size, as large perhaps as tangerine oranges, or smaller multiple nodules may be disseminated through the lungs. They are usually soft, rounded, and well defined, and present a fleshy appearance.

The pulmonary lymphatics are often infiltrated in the neighbourhood of the growths, and in some cases of cancer the growth infiltrates the subpleural lymphatics, and appears beneath the pleura as flattened masses.

Secondary tumours are more often multiple and rounded; epitheliomata, for example, may occur as round masses completely studding both lungs, but a well-defined single mass may be present. In rare cases the growths are so small and widely disseminated as to simulate tubercle, and in some cases of this nature each nodule presents a shiny white appearance.

Secondary deposits in other organs are occasionally met with in cases of primary cancer of the lungs.

With widely disseminated multiple growths the lungs are usually enlarged, and a dense infiltration may have a similar effect; but the latter lesion is not uncommonly associated with some degree of retraction.

If softening of the growth takes place, cavities form, and collections of pus may be produced either in this manner or from suppurative changes in the neighbouring tissue.

When a primary growth is limited to a single lung, the right side is more often affected than the left. Secondary tumours, as already stated, are usually multiple, and affect both lungs.

Etiology.—*Age.*—Primary malignant disease may occur at any age, but is most common in later middle life.

In 136 cases of cancer collected by Walshe, 57 were fatal between the ages of forty-one and sixty.

Sex.—The incidence of the disease on the two sexes is, according to Walshe, nearly equal.

Heredity.—It has been pointed out by Roger Williams with regard to cancer generally that it is more common in cases of that disease to find a family history of tuberculosis than one of malignant disease. We are not aware whether this point has been worked out with regard to primary cancer of the lungs.

Symptoms.—So long as the growth is confined to the lung, and does not affect the mediastinum, it may give rise to few if any symptoms suggestive of its presence; and it is by no means uncommon for a growth to be discovered after death in a case which had possibly been regarded as one of bronchitis. This has occurred in the majority of cases of enchondroma and other rare tumours already mentioned. An enchondroma may, however, cause occlusion of a bronchus. The diagnosis of a mediastinal tumour is generally made from the pressure effects to which its presence gives rise, but, so long as a growth is limited to the lungs, these effects are necessarily absent.

The *symptoms* are such as are common to a variety of chronic pulmonary diseases—namely, dyspnœa, cough, expectoration, hæmoptysis, emaciation, and pyrexia. The physical signs more often suggest the presence of bronchitis or consolidation of the lung than that of a tumour. The dyspnœa may, however, be out of proportion to the physical signs, and the expectoration has in some cases the peculiar red currant jelly or prune juice character.

A rapid dissemination of cancer throughout the lungs has been known to be attended by acute symptoms suggestive of pneumonia—*e.g.* rigors, labial herpes, severe pyrexia, consolidation of the lung, and albuminuria (Wunderlich). In other cases pyrexia, sweating, and emaciation have been present. A hæmorrhagic pleural effusion may also occur.

Physical signs.—The extreme rarity of primary growths confined to the lungs, and not involving any of the structures in the mediastinum, must be held to justify a somewhat brief consideration of the signs by which their presence may be attended.

Inspection.—When a growth has attained a large size local bulging may be observed, but this is by no means invariable, as retraction may result from an infiltrating growth, or from collapse secondary to bronchial obstruction.

Expansion is diminished according to the size of the growth and its situation. With widely disseminated growths the intercostal spaces may recede during inspiration.

Palpation.—Vocal fremitus is often retained and may be increased, unless the growth is situated beneath the chest wall (which is rare) or has obstructed a large bronchus. The cardiac impulse may be displaced, and pulsation may be produced by the movements of the heart being transmitted through the solid growth in the lung to the chest wall.

Percussion.—The percussion note may be unaltered, or absolute dulness may be present. It may also undergo various modifications, according to the extent of the growth and the condition of the lung intervening between it and the chest wall. Extension of the dulness beyond the middle line has the same important significance in the case of a growth limited to the lungs, as in mediastinal tumour, but it is naturally of less common occurrence.

Auscultation.—It may be stated generally that all, or nearly all, the alterations in the breath sounds and voice sounds which characterise a mediastinal growth may be present in cases of tumour limited to the lungs, and the same holds good of the adventitious sounds which may be audible. These are, as already stated, indicative of physical conditions which may be produced by either lesion.

Diagnosis.—If in the course of its enlargement the growth extends to the mediastinum, the diagnosis, previously perhaps almost impossible, may become a matter of but little difficulty; failing this, its presence is very likely to be overlooked.

The possible presence of a secondary growth in the lungs should be borne in mind when pulmonary symptoms appear after the removal of a cancer or sarcoma, or when the growth is still *in situ*. More than ordinary care should be taken in such cases before a diagnosis of 'bronchitis' is made, and the special liability of the lungs to be the seat of secondary sarcomatous growths must be remembered.

If, as we have repeatedly urged throughout this work, an examination of the expectoration for tubercle bacilli be made as a matter of routine in all cases of pulmonary disease, and if a destructive lesion is present but no bacilli are found, the diagnosis will by a process of exclusion be facilitated.

Reference may be made to the chapter on Mediastinal Tumours for a more detailed discussion of some points in the diagnosis of morbid growths within the thorax.

Duration—Prognosis.—In cases of malignant disease a fatal termination is inevitable. The duration of the disease varies within nearly the same limits as in mediastinal tumour. Walshe gives the maximum as 27 months, the minimum as 3 months, and the average as 13·2 months.

Treatment.—The symptoms which cause most distress must be relieved as they arise by measures similar to those suitable in cases of mediastinal tumour.

For observations as to surgical treatment, see below.

J. K. F.

REMOVAL OF PORTIONS OF THE LUNG OR PLEURA FOR TUMOURS AND OTHER MORBID CONDITIONS

It has been shown experimentally by Gluck, Schmidt, Block, Biondi,[1] and others that considerable portions, and indeed the whole of the lung, may be removed without necessarily causing the death of the animals operated upon. When portions only of the lung were removed, the pneumothorax disappeared in a short time, the remaining lung enlarging or becoming displaced to occupy the cavity which was left. If the whole lung was removed, it must be presumed that a permanent pneumothorax remained.

---

[1] Gluck: 'Experimenteller Beitrag zur Frage der Lungenexstirpation.' *Berl. klin. Wochenschrift*, 1881, Nr. 44, p. 645.

Hans Schmidt: 'Experimentelle Studien über partielle Lungenresection.' *Berl. klin. Wochenschrift*, 1881, Nr. 51, p. 757.

Block: *Deutsche med. Wochenschrift*, Nr. 44, 1882, and *Verhandlungen der deutschen Gesellschaft für Chirurgie*, 1882, p. 77 ff.

Biondi: *Wiener med. Jahrbücher*, 1882, Heft 2-3, 'Lungenexstirpation bei experimentell-localisirter Tuberculose.'

On the strength of these experiments, numerous operations have been undertaken for the removal of portions of lung affected by tuberculosis. Mr. Stephen Paget, in his valuable book on the surgery of the chest, has given an interesting *résumé* of the subject, in which it appears that, while several of these operations have proved fatal, two at least have been successful. One was undertaken by Mr. Lowson,[1] in which the tubercular apex of the right lung of a woman, aged thirty-four, was removed after resecting portions of the second and third ribs in front, the patient dying, probably of gastric ulcer, nine months after the operation. The other case was that of a young man of nineteen, also with tubercular disease of the right apex. He was under the care of M. Tuffier, who in 1891 removed the affected part through an incision in the second intercostal space without resecting any portion of rib. The patient was in good health four years later.

With regard to these operations it must be said that they do not seem likely ever to have a very wide application; not only because it is very difficult to be sure that the part on which it is proposed to operate is the only one affected, but also because, in the early stage in which alone surgical interference would be justifiable, there is a fair chance of effecting a cure by other less hazardous measures. The larger the portion of the lung to be removed, the more dangerous naturally will be the operation; and it is hardly to be expected that the total extirpation of one lung for tuberculosis will ever be considered a justifiable proceeding, considering the very great probability that the other either has been already or will at some future time be affected with the same disease.

It may be argued on the other side that one tuberculous kidney has often been removed with excellent results. But to this it may be answered that it is much more common for one kidney only to suffer than for one lung only to be affected by tuberculosis, and also that the records of the removal of tubercular kidneys are not so free from disaster as to warrant, in the opinion of many surgeons, indiscriminate nephrectomy in this disease, except for the removal of very troublesome symptoms.

The total extirpation of the lung has also been recommended for another condition, namely—where, in a case of empyema, after extensive 'Estlander's' operations, obliteration of the pleural cavity has been effected, but the patient is left with a shrunken fibroid lung riddled through and through with bronchiectatic cavities. The disappointment both of the surgeon and the patient under such circumstances must be great; but I have not heard of such an operation being actually performed, although it has certainly been contemplated. The danger of removing the organ, bound down as it must be to the structures in the mediastinum by firm adhesions, and with the normal relations of the parts disturbed by the contraction that has been going on, cannot fail to be very great.

---

[1] *British Medical Journal*, 1893, vol. i. p. 1152.

The subject is not yet sufficiently matured to enable one to lay down any definite rules of procedure in the operation. In removing a portion only of the lung, it has been recommended (Tuffier) to avoid making a pneumothorax by stripping off the pleura from the ribs for some distance below the point at which it will ultimately be necessary to open it, and suturing the two layers together before incising it. A sort of partial pneumothorax, it is said, is thus established outside the pleura. Tuffier, as has been mentioned, did not remove a portion of rib, but others have removed portions of the second and third, or the second, third, and fourth ribs.

For the removal of the whole lung a large flap and a still greater resection of ribs would be required. In the experiments on animals a ligature was placed round the whole root of the lung.

The so-to-speak accidental removal of portions of the lung is quite another matter. Nature herself occasionally removes a considerable mass of pulmonary tissue by a process of necrosis, and the sphacelus may be found floating in the pus of an empyema. Reference has also been made to the removal of a portion of lung in cases of *hernia pulmonis* without any detriment to the patient (p. 498). But the question sometimes arises practically in the course of removal of tumours of the chest wall which have invaded the pleura and the lung. It is often quite impossible to ascertain beforehand with any degree of certainty how deeply such tumours have extended; and if the surgeon should find that the lung is invaded by the growth, he should not at once give up the operation as hopeless.

The right course under such circumstances is to ascertain as far as possible with the finger the extent to which the lung is involved, and this examination should not only include the immediate neighbourhood of the parietal tumour, but as much as possible of the rest of the lung should be explored for secondary growths. If the mass should not appear very extensive, an attempt should be made to remove it. This has been successfully accomplished on several occasions, sometimes by means of the actual cautery, sometimes by cutting it away after placing ligatures beyond its limits, and in some instances by simply snipping it across with scissors. Occasionally it has been necessary to leave pressure forceps attached to bleeding points which it was impossible to secure by ligature. I have never myself had to deal with such a case, and have only once assisted at an operation of the sort, which occurred some years ago, and in which it did not seem wise to attempt the removal of the growth. It appears to me, however, that the use of the ligature is the fact that the lung is bound down to the chest wall by adhesions opposite the spot where the pleura is wounded, or that the lung has been caught between the fragments of a broken rib. Air then continues to escape during the act of expiration until the wound in the lung is closed, which usually occurs in the course of a few hours. The condition, though very disfiguring, seldom causes any serious symptoms, and the air is absorbed in the course of a few days. If necessary, its escape may be brought about by making punctures in the skin, but this is seldom necessary, and if not required is unadvisable.

Pneumothorax is sometimes a consequence of wound of the lung. A non-adherent pleura is essential for the development of this condition. The wound may be small or large. Pneumothorax is a much more serious complication than general emphysema. It is detected by the characteristic physical signs (*vide* p. 636). Here again the wound in the lung is usually rapidly closed, probably by the formation of a clot, and then the air in the pleura is absorbed in the course of a few days.

Sometimes, however, probably when the wound in the lung is valvular and large, air continues to escape into the pleura, considerable pressure is produced, the lung collapses and is compressed, and the mediastinum is pressed over to the opposite side. In such a case surgical interference is urgently required. Fortunately it is simple and efficient : a cannula and trocar of small size are introduced through an intercostal space, and the cannula is left *in situ*. It is necessary to be as careful about the antiseptic arrangements for this simple operation as for any other, or a septic pleurisy may be set up under most unfavourable circumstances. As long as the air continues to escape from the lung the cannula must be retained, and it must be covered by an efficient antiseptic dressing. It is sometimes difficult to be sure whether or not the wound in the lung is closed. If there be any doubt, the cannula must be removed and a careful watch maintained. Should it be evident that the air is accumulating in the pleura, the cannula must be again introduced.

Bruising of the lung.—This is a frequent accompaniment of fractured ribs, and if not great in amount is a matter of very little moment. When a single rib or not many ribs are fractured, it is not uncommon for a few pellets of dark blood mixed with mucus to be expectorated for a fortnight or more after the accident. Sometimes the hæmoptysis does not begin immediately, perhaps even not till the second or third day. It is probable that this depends upon the bruising of the lung, though it is not possible to exclude the presence of laceration.

When the injury to the chest wall is more extensive, the hæmoptysis may be expected to be more severe.

Usually no physical signs are obtainable in mild cases. But sometimes pneumonia may follow either slight or severe contusions, and this is one of the dangers to which old people especially are subjected after fracture of ribs.

Laceration of the lung.—This may occur either as the result of fracture or simple bending of the ribs, or as the result of penetrating wounds of the chest, and is generally, if not always, accompanied by more or less severe hæmoptysis and hæmothorax; but if there be an external wound there may be free external hæmorrhage, and if the lung be adherent to the pleura this hæmorrhage may completely take the place of the hæmoptysis and hæmothorax. Some of these injuries may be followed by hernia of the lung, which will be considered later.

The lacerations resulting from fractured ribs are usually to be treated simply by keeping the patient at absolute rest; but in desperate cases, where the condition has been diagnosed, it may be advisable to open the chest, and to endeavour to arrest the bleeding either by plugging the wound in the lung or by bringing the edges together by means of sutures. The cases must, however, be very rare in which such a proceeding is desirable.

Injuries of the lung from external wounds are not common in civil practice in modern times, but the writings of older surgeons teem with examples of those who sustained such injuries either in duels or at the hands of assassins; and no better illustration of the change of opinion that has taken place in the matter of treatment can be gained than by reading the accounts of people who were stabbed, and in whom copious hæmoptysis or external hæmorrhage was the sign for the surgeon to let blood from the arm. It is quite remarkable, too, to notice how many recoveries are reported; probably the fatal cases were not thought worthy of permanent record. At the present day we meet with a certain number of machinery accidents and a few cases of accidental or suicidal gunshot wounds or stabs, and in military practice such wounds are of course still common enough.

In cases of *bullet wound* it is very unlikely that much or anything will be gained by a minute exploration of the wound, but there can be no objection, after a most careful cleansing and rendering aseptic of the external parts, to the gentle introduction of a purified probe or the finger into the wound, provided that no very extensive examination be made if nothing is found. The patient will have a better chance if he be kept absolutely at rest in the recumbent posture. It is very likely that some pneumonia will supervene, but it does not follow that it will be severe; and if no portion of the clothes or other septic material has been introduced with the bullet, the patient may very likely escape septic inflammation of the lung. It is highly probable that this department of surgery may be much aided by the use of the X rays, but this investigation will be most safely made when the patient has recovered from the immediate effects of the injury.

Hernia of the lung.—This is a subject on which I must write without any personal experience. I have often been asked to see patients supposed to be suffering from hernia of the lung, but with one doubtful exception they have all turned out to be cases of abscess coming from within the chest, some containing pus only, others pus and gas. The former differ from hernia of the lung in yielding a more or less dull percussion note, but breath sounds are often very distinctly heard over them. The latter, on the other hand, have a tympanitic percussion note, but little or no breath sound is to be heard over them. Such abscesses usually start from disease of some part of the bony framework of the thorax, or are the pointings of empyemata, but they may arise from a variety of causes; they ought not often to give rise to any serious difficulty in diagnosis.

It will be convenient to discuss the whole subject of hernia of the lung together, and to clear the way by a short description of **spontaneous or non-traumatic hernia.** The existence of this depends either on an unusually high position of the apex of the lung or upon some natural defect of the chest walls. The former condition has given rise to a prominence in the posterior triangle, which has a marked impulse on coughing, and it is conceivable that such a prominence may be caused or, if present, aggravated by prolonged cough. It was a case of this sort which I mentioned as the doubtful exception to my complete personal ignorance of the subject; and in this case I could not convince myself that the swellings in the posterior triangles did not consist of a local hypertrophy of the fatty tissue of the part. It is obvious that soft tumours in this situation, such, for example, as the fatty tumours met with in sporadic cretins or the swellings caused by cystic hygroma, might very easily be mistaken for protrusions of the lung. But there can be little doubt that genuine cases have been occasionally met with.

If hernia occurs in connection with congenital defect of the chest wall, the nature of the case is likely to be quite obvious. But, as a matter of fact, congenital hernia of the lung is so excessively rare that only one or two cases are met with in medical literature. There is a celebrated case of a monster described by Cruveilhier ('Anatomie pathologique du Corps humain,' vol. i. livraison xix., Pl. VI., 1829–1835) in which the right lung extended far up into the neck; and there are others probably depending upon some injury at the time of birth, which therefore ought to be classed amongst the traumatic cases.

In ectopia cordis the lung does not project with the heart. In the cases which I have seen of congenital deficiency of parts of the sternum and ribs, so far from a hernia of the lungs occurring, an actual recession of the organ was observed.

This part of the subject need not therefore detain us further.

Traumatic hernia of the lung.—Traumatic hernia of the lung results from some injury of the chest walls with or without open wound, and it may occur at the time of the accident or follow it at some considerable interval. To the latter condition the term **Consecutive hernia** has been applied.

The accident that produces consecutive pulmonary hernia without external wound usually involves a fracture of one or more ribs. For some unexplained reason the chest wall remains soft at the point of injury, and the effort of coughing causes a portion of lung to be extruded at the weakened part, which sometimes only makes its appearance at the time of forced respiratory efforts such as those of coughing or defæcation, but which sometimes becomes permanently engaged in the opening. In the latter case, as in others to be presently described, the extruded portion of lung enlarges, owing probably to its vesicles becoming emphysematous, and so forms a rounded tumour attached to the lung itself by a pedicle the size of which corresponds to the size of the aperture. A few such cases have been recorded, such, for example, as one by Dr. Joseph Bell,[1] where a baby thirteen months old had a tumour as large as a hen's egg three inches below the right axilla in the axillary line. But it is a remarkable fact that while fracture of ribs is one of the commonest of accidents, the condition we are describing is one of the rarest of surgical rarities

Consecutive hernia of the lung following an open wound of the chest which has healed differs in no way from the form we have just been considering, except that here there is a scar which gives way in the same manner as happens in the formation of ventral herniæ after laparotomy. Here again one cannot help observing that, although numberless cases have been observed where, after the removal of portions of rib in the treatment of empyema, no regeneration of the bone has taken place, there is no record that I am aware of one in which a hernia of the lung has followed. Probably the collapsed or consolidated state of the lung and the presence of pleural adhesions have much to do with the immunity that certainly exists from this accident.

Much discussion has taken place as to how it is that the hernia is ever produced. It is pretty clear that it can only be caused by expiratory efforts with a closed glottis; and it has been held by some that an emphysematous state of the lung is an essential part of the condition. Without more personal experience it is only possible to state the problem without attempting to supply an answer.

The *diagnosis* of these cases is made by noting that the tumour is rounded and soft, more or less reducible, and imparts to the hand a characteristic crackling sensation, such as is felt on handling a portion of healthy lung. It is resonant on percussion, and over it breath sounds are distinctly audible. There is an impulse on coughing—*i.e.* during expiration—not, as has been stated by some, during inspiration. This latter statement must have been made as the result of imperfect observation. It is most easily confused with a nævus or other very vascular growth.

The *treatment* may be radical or palliative. If the hernia causes no inconvenience it may be left alone, or a pad may be worn to prevent the protrusion. If a complete cure be desirable the tumour may be cut down upon, and after placing a ligature round it the protruding portion may be cut off, and then the opening in the chest wall may be closed by sutures. It seems probable that in some cases the protruding portion can be simply returned through the opening, which may then be closed as in an ordinary operation for the radical cure of hernia.

---

[1] 'Five Years' Surgery in the Royal Hospital for Sick Children,' *Edinburgh Hospital Reports*, 1893, p. 471.

Hernia of lung accompanying an external wound. There are many cases recorded where the lung has protruded through an incised wound of the chest wall. The injury is mostly in front, the seventh, eighth, and ninth interspaces near the junctions of the ribs and their cartilages being, so to speak, the seats of election. The *rationale* of this accident is somewhat difficult to understand. I imagine that it must result from a violent expiratory effort, with closed glottis, at the time of injury. This forces a piece of the lung into the opening, where it is gripped between the ribs and prevented from returning. The accident usually happens when there is no injury to the lung, but it may be accompanied by a wound of the organ. The reason is obvious. A wound of the lung of any size not only encourages collapse, but, from the very fact of the escape of air from its surface, tends immediately to separate the visceral from the parietal surface of the pleura.

There is not likely to be any difficulty about the diagnosis of such a condition. The protruded lung soon becomes very dark in colour, and, it is said, may pass into a state of gangrene. The presence or absence of hæmoptysis depends upon the existence or not of a wound of the lung.

The *treatment* may consist either in returning the lung after the most careful attempt to render it aseptic, and the closing by sutures of any wound that may be seen; or, if it appears very unhealthy, a ligature may be placed around the base of the protruding mass and it may be removed; after which the wound, if it be clean, may be closed.

The subject of hernia of the lung, partly perhaps from the physical problems involved, partly from its great rarity, has excited a good deal of interest. The reader who is desirous of pursuing the matter further is referred to a very exhaustive treatise by Morel Lavallée,[1] and to a shorter article by Strübing.[2] Mr. Stephen Paget[3] has also a very interesting chapter on the subject.

Contusio and commotio thoracis.—In concluding this subject some mention must be made of the conditions described under the above titles.

By *contusion of the chest* is meant any injury to the intrathoracic viscera with or without injury to the chest wall. Much, therefore, of what has already been described would have to be included under this heading. The enormous amount of injury that may be done to the organs with little or no injury to the ribs is truly remarkable. This, of course, is especially likely to happen in children and young people in whom the ribs are very flexible. I described a case in the 'Pathological Transactions' for 1875, vol. xxvi. p. 13, where a boy seven years of age was run over by a cart wheel, which only broke the third, fourth, and fifth ribs at their greatest convexity. The right lung was adherent to the chest wall, the left was free from adhesions, and neither had sustained any serious injury beyond bruising; but the trachea was ruptured by a vertical split both in front and behind, and there was extensive hæmorrhage in the mediastinum in consequence.

---

[1] 'Hernies du Poumon.' *Mémoires de la Société de Chirurgie de Paris*, tome premier, 1847, p. 75.

[2] 'Ueber spontane Lungenhernien der Erwachsenen,' Virchow's *Archiv f. path. Anat. und Phys. und f. klin. Med.*, 1889, Band 116, p. 205.

[3] *The Surgery of the Chest*, 1896.

About contusion of the chest I need not say more. It can hardly exist without some of the obvious diagnostic symptoms previously described.

Commotio thoracis implies that a blow or injury to the chest produces symptoms for which no adequate organic lesion is discovered. The term reminds one of the discussion as to whether *concussion* can result from an injury to the head which produces no organic lesion of the brain. The fact of its existence has been carefully noted and elaborately accounted for by some observers, and denied by others. That a sharp blow on the sternum or some other part of the chest may give rise to immediate and very serious symptoms, notably feeble pulse, diminished blood pressure, rapid and shallow breathing, pallor and cold sweats—in fact, to all the symptoms of shock—and that the patient may recover without any other symptom attributable to injury of heart or lungs or other organs is beyond dispute; but the problem is so complicated by the possibility of other concomitant injuries that it must be very difficult to estimate the actual part that is played by the shaking up of the intrathoracic viscera in the production of the effect. Numerous experiments have been made upon animals with the view of ascertaining how much depends upon injury of the pneumogastric and the sympathetic nerves, but the mere fact that they seem to prove that it is in this direction that we must look for an explanation of the symptoms, makes it less necessary to pursue the subject further in discussing the question of injuries of the lungs and pleura.

R. J. G.

CHAPTER XLIV

DISEASES OF THE PULMONARY VESSELS

EMBOLISM OF THE PULMONARY ARTERY

	PAGE
Embolism: Etiology	500
Characters of emboli	501
Effects of embolism	502
Pathology of infarction	503
Symptoms	505
Physical signs	506
Diagnosis	506
Prognosis	506
Treatment	507

Etiology.—1. *Detachment of a thrombus in a systemic vein.*—A thrombus having formed in some part of the venous system becomes detached, and, passing through the right cavities of the heart, blocks either the trunk of the pulmonary artery or one of its main branches or a secondary branch. This may happen in the puerperal state from dislodgment of a thrombus formed in the uterine, ovarian, or pelvic veins; in thrombosis of the femoral and saphenous veins from whatever cause arising; in thrombosis of the lateral sinus and jugular vein, secondary to disease of the middle ear; in marasmic thrombosis occurring in the course of anæmia or pulmonary tuberculosis; also in typhoid fever, pleurisy, and various septic affections.

2. *Detachment of a cardiac thrombus.*—If a thrombus forms during life in the right auricle or its appendix, or in the ventricle, and becomes detached, it passes into the pulmonary artery and lodges in the first branch of the vessel which is too small to allow it to pass.

Asystolism of the right ventricle from any cause may be accompanied by the formation of clots in the recesses of the columnæ carneæ, and by rounded clots at the apex or elsewhere. Vegetative endocarditis of the tricuspid or pulmonary valves is a rare condition antecedent to pulmonary embolism.

3. A portion of a *parietal thrombus in the pulmonary artery* may be washed away and become impacted in a branch of the vessel more distant from the heart. This event may occur in various pulmonary affections, but it is not often observed.

4. *Fat embolism.*—The capillaries of the pulmonary artery may be blocked by minute portions of fat which have gained access to the vessels. This is usually the result of fracture of a bone, or of extensive injury to the subcutaneous tissue or of rupture of the liver. In some cases of diabetes, fatal from coma, fat embolism has been observed in association with the condition known as 'lipæmia.'

In fat embolism the capillaries of the pulmonary artery may be shown to contain in certain areas homogeneous masses of oil which, on staining with perosmic acid, appear as a black network of vessels upon the walls of the alveoli. If this change is sufficiently extensive it materially obstructs the passage of blood through the lungs and impedes respiration.

5. A *new growth* may perforate a systemic vein, and minute portions may become detached and plug the pulmonary capillaries and give rise to secondary growths in the lungs.

6. *Air embolism.*—Fatal symptoms may quickly follow the admission of a considerable quantity of air in a short time into the venous system, but the repeated admission of small quantities of air is not usually attended by serious results. The pathology of this condition is doubtful; it has been attributed to embolism of the pulmonary capillaries; but of this there is no absolute proof. According to Hamilton[1] it is due to the fact that the air, being lighter than the blood, cannot circulate through the capillaries. The post-mortem appearances in such cases show that the air is churned up with the blood in the right ventricle of the heart, which becomes enormously dilated. It is probable, as suggested by Coats, that death follows from the inability of the heart to drive the blood into the lungs, owing to the force of the systole being expended in compressing the air in the ventricle, and that during diastole the compressed air expands and dilates the cavity. Such a condition must also obstruct the entrance of blood from the venæ cavæ.

7. In rare cases *Hydatids* have gained access to the circulation, and branches of the pulmonary artery have been obstructed by secondary cysts.

8. A *phlebolith* formed in a vein has been known to pass into the circulation and become lodged in a branch of the pulmonary artery.

Characters of emboli. The clot, if of considerable length, is very likely to be doubled upon itself and may be lodged at the bifurcation of the main vessel, or it may plug one of the primary branches, or, if smaller, it may be found astride the angle formed where a branch is given off.

A thrombus dislodged from a vein will usually present the stratified appearance characteristic of a slowly formed clot; it consists of fibrin, enclosing white and some red corpuscles in its meshes and is paler in colour, more firm and dry than the clot which is often found around it, which is either of ante- or post-mortem formation. If a site of origin can be discovered the embolus may be found to correspond exactly to the size of the vessel, and at the point of separation to the ragged end of the clot still *in situ*. If death has followed immediately upon its impaction, it will be found lying free, generally about the bifurcation of the vessel; but if it is impacted in a smaller branch and time allows, the clot becomes adherent to the wall, and secondary thrombosis occurs. The newly formed clot extends towards the heart, and very often to a slight degree in the opposite direction also; in either case it tails off towards its extremity. In old lesions the wall of the obstructed vessel is found thickened and fibrous or atheromatous, and possibly dilated beyond the site of obstruction, or the clot may be tunnelled. If the clot is derived from a septic focus, suppurative changes may be set up in the walls of the blocked vessel, and the secondary thrombus may break down.

Coagula derived from the cardiac cavities or vegetations from the valves can rarely be recognised by their correspondence to clots still *in situ*, but careful search should be made for such clots in the right auricular appendix, and the recesses of the columnæ carneæ. Rounded fibrinous balls with smooth exterior and softened grumous contents are sometimes formed in these sites and may become detached, but they are more frequently met with in the left cavities of the heart.

Emboli tend especially to plug the branches of the vessels distributed to the lower lobes.

Effects of embolism.—These depend chiefly upon the size of the vessel obstructed, and the septic or aseptic character of the embolus. If a small branch is blocked by an aseptic embolus, transitory collateral hyperæmia may be the only result. Blocking of larger branches or extensive capillary embolism is generally accompanied by œdema.

In rapidly fatal cases the lungs may be found over-distended with air and œdematous, or collapse limited to certain areas may be present with distension of the surrounding tissue.

When the embolus is derived from a septic source, inflammation occurs not only in the walls of the obstructed vessel, but also in the lung tissue around, and a pneumonic infarct is formed. The infarct is usually situated on the surface of the lung, and presents a wedge-shaped outline; but it is not always so limited. Consolidation is thus produced, and the part quickly assumes the appearances of grey hepatisation; it may then undergo complete necrosis and break down, an abscess cavity resulting. Gangrene of the consolidated area is also occasionally observed to follow the lodgment of a septic embolus.

In septic cases the pleura may also become infected, usually in areas which correspond to the site of pyæmic infarctions. Acute inflammation follows, and may be accompanied by a fibrinous, serous, purulent, or hæmorrhagic effusion. Perforation of the pleura covering a septic infarction is one of the rarer causes of pneumothorax.

The lesion which is usually described as typical of aseptic embolism of the pulmonary artery is the **hæmorrhagic infarction** (pulmonary apoplexy). There is great difference of opinion as to the pathology of this condition, and the only point upon which all observers are agreed is that it is a form of pulmonary hæmorrhage.

A hæmorrhagic infarction is similar in some respects to those found in the spleen and kidney in cases of vegetative endocarditis. When recent, it is of a deep purple red colour, sharply defined, solid and smooth on section, and is wedge-shaped, with the apex at the point of obstruction, and the base projecting above the pleura. In the majority of cases it extends to the surface, but if near the root or in the interior of the lung it may be rounded and is then usually of smaller size. The branch of the pulmonary artery leading to it will be found blocked by a thrombus in which, in some cases, the original embolus can be found. Infarctions vary greatly in size; they may be no larger than a pea, but generally they are about an inch or two across at the base. The lesion may however involve the whole of a lobe.

A specimen in the possession of the writer shows complete consolidation of the middle lobe of the right lung and partial consolidation of the lower lobe. In cases of mitral stenosis, in which the lesion is very common, several infarctions are often present.

The overlying pleura is usually inflamed, and either presents minute extravasations or may be covered with fibrinous exudation. When of older date infarctions lose their deep purple colour and become of a brownish red tint. At a still later period they contract, and their site may be ultimately marked by a pigmented area in which the texture of the lung appears coarse and fibrous. Bronchial dilatation may occur in the neighbourhood of such a lesion. Possibly, in cases of valvular disease, the blood in a small infarction may be expectorated and the circulation may be ultimately re-established through the obstructed vessel. On *microscopical examination* of a pulmonary infarct the bronchioles, alveoli, and pulmonary capillaries are seen to be distended with red blood corpuscles; some catarrhal cells are also present in the alveoli, due to the irritation set up by the presence of the blood, and there may be slight traces of fibrin formation.

For the formation of an infarction a certain time is necessary, probably from two to three days; for this reason they are not met with in cases which are quickly fatal.

Pathology of infarction.—The exact method by which the blood obtains access to the area supplied by the obstructed infection, the temperature being of a hectic type, with profuse sweating, marked prostration, and perhaps diarrhœa. If the lung breaks down, the sputa may be chocolate coloured from admixture with blood, or the contents of an abscess cavity may be expectorated. If perforation of the pleura occurs, pneumothorax and a most acute inflammation of the pleura follow, and the air contained in the pleural cavity may quickly acquire a fœtid odour.

Physical signs.—In severe cases, if an examination is possible, the inspiratory sound will be found to be harsh and expiration somewhat prolonged. Pneumonia, if present, will be attended by the ordinary signs; but it must be remembered that crepitation, with dulness on percussion, and tubular breathing, and a pleural friction sound may accompany the formation of an infarction. Œdema will give rise to fine crackling râles. If collapse occurs the breath sounds will be absent over the corresponding area. There may be signs of dilatation of the right cavities, and a systolic murmur is often audible over the pulmonary artery. This may subsequently disappear if recovery takes place.

Diagnosis.—In a case in which venous thrombosis is known to exist, the sudden onset of dyspnœa, presenting the characters already described, and accompanied by intense agony should at once suggest the nature of the condition.

In obstruction of the glottis air enters the chest with difficulty, whereas in embolism of the pulmonary artery the patient feels that he cannot breathe too deeply.

Asthma is accompanied by sonorous and sibilant rhonchi and by far more marked prolongation of the expiratory act.

We can recall a case of valvular disease seen when the patient, just admitted to hospital, was almost *in extremis*, in which the presence of tubular breathing over a large area at the base led to the diagnosis of pneumonia. This was proved post-mortem to be erroneous, the consolidation being due to a very large hæmorrhagic infarction, which occupied the greater part of the right lower lobe.

Prognosis.—It is obvious from the above description that the condition under nearly all circumstances necessitates a grave prognosis. If the patient is seen after recovery has followed from the alarming symptoms of the onset, the prognosis will partly depend upon the source of the embolus; if it is of septic origin, pneumonia, pulmonary abscess, and pneumothorax are very likely to follow and to terminate fatally. Non-septic emboli of small size are much less dangerous, but the possibility of a fresh dislodgment of clot and of more extensive blocking of the vessel by the formation of thrombi at the site of impaction of the emboli must be borne in mind. Extensive fat embolism of the capillaries is a very dangerous condition.

In valvular disease of the heart hæmorrhagic infarction more often occurs towards the close of life than during periods of fairly good health, but it may occur quite apart from signs of cardiac found in a large branch of the pulmonary artery, and several infarctions in the corresponding lung, but none in the other lung. In such cases it appears extremely probable that portions of the thrombus have been washed off, forming emboli which have blocked smaller branches and have led to infarction.

In cases in which an embolus cannot be identified within the thrombus, or in which no obvious source of an embolus is discovered, it is at least conceivable that the thrombus within the branch of the pulmonary artery leading to the infarct is the primary lesion, and that the infarct has formed after the obstruction of the vessel has become complete. Whilst, therefore, we consider that the question is still an open one, we are not disposed to adopt the view that the wedge shape of the infarct is due to the shape of the terminal bronchus and attached air vessels, and that the formation of a hæmorrhagic infarction in the lungs is entirely unconnected with vascular obstruction.

Symptoms.—In the rapidly fatal cases, in which the trunk or a main branch of the vessel is obstructed, the onset of the attack is sudden, the patient being seized with intense dyspnœa and pain in the chest. There is great agony and fear of impending death; the action of the heart is either tumultuous and irregular or extremely feeble, and the pulse may disappear. The superficial veins are distended, the lips blue, and a clammy sweat breaks out. In such cases death may occur within a few minutes, either from asphyxia with convulsions, or from syncope due to cardiac paralysis.

If the patient recovers from these very urgent symptoms rapid breathing of a panting character continues. Air enters the chest freely, but this affords no relief to the feeling of want of breath. The heart may now be acting quietly. If life is prolonged the rapid breathing continues, or Cheyne-Stokes respiration may occur. After a day or two blood may appear in the expectoration. The temperature is usually low. Exophthalmos is present in some cases. Death may take place in a few days from fresh embolism or extending thrombosis, but recovery has been known to occur in cases which at the outset appeared hopeless.

Capillary embolism, if extensive, may be accompanied by urgent dyspnœa, ending quickly in death from asphyxia, or the process may be less rapid, the degree of dyspnœa gradually increasing with the increase in the area affected.

If the vessel blocked is of smaller calibre the symptoms will be much less severe. In cases of mitral disease pain in the side, followed by hæmoptysis, or the expectoration of dark blood-stained mucus are the symptoms which usually point to the occurrence of infarction, and in some cases the exact site of the infarction is indicated by a localised pleural friction sound, and tenderness on forcible percussion over the same area (Head[1]).

If the embolus is infective and pneumonia or abscess follows, rigors generally occur, followed by all the symptoms of septic

CHAPTER XLV

THROMBOSIS OF THE PULMONARY ARTERY

	PAGE
Ætiology	508
Spontaneous thrombosis	509
Organisation of a thrombus	510
Effects of thrombosis	510
Symptoms	510
[illegible] of thrombosis	511
Diagnosis	512
Treatment	512
Atheroma and sclerosis of the pulmonary artery and veins	512

Ætiology.—Thrombi may be formed during life in the pulmonary artery under a variety of conditions, of which the following are those of most frequent occurrence.

(1) During the process of dying, particularly in cases where this is prolonged, a thrombus may form at the apex of the right ventricle, and become united through the tricuspid orifice with a similar clot starting in the appendix of the right auricle. It then extends through the pulmonary orifice, where it is so moulded by the cusps of the valve that three winged projections form upon its surface. Thence it may be continued into the branches of the pulmonary artery to its most minute ramifications. In other cases a clot of a similar character originates behind the cusps and spreads into the artery. A thrombus of this nature consists of firm, pale, tough fibrin ; it is not attached to the walls, and can be pulled out whole from the vessel, the lumen of which it does not completely fill.

(2) In various pulmonary diseases, such as pneumonia, tuberculosis, cirrhosis of the lung, and gangrene, the branches of the pulmonary artery distributed to the affected part may be found filled with adherent fibrinous coagula. In such cases the clotting is probably due to changes in the vessel wall induced by the neighbouring disease. In tubercular cases it may result from an extension of the process to the coats of the artery.

(3) In a hæmorrhagic infarction the branches of the pulmonary artery in the infarcted area almost invariably contain thrombi (*vide* p. 504).

(4) After embolism of the pulmonary artery from any cause, if life is sufficiently prolonged, secondary thrombosis almost invariably occurs on the cardiac side of the obstruction.

(5) Atheroma of the artery, or changes in its coats, the result of the pressure of an aneurysm or a tumour, may be followed by the formation of a parietal thrombus, which may by gradual increase become obliterative.

(6) **Spontaneous thrombosis.**—The grave symptoms described as characteristic of embolism of the pulmonary artery (*vide* p. 506) are admitted by all writers to be commonly due to that cause. Some, however, believe that similar symptoms may arise from the spontaneous formation of a thrombus in the vessel, and that sudden death may thus result. This view has not yet met with general acceptance, as no satisfactory cause has been assigned for the occurrence of thrombosis, independently of any of the conditions already mentioned, except that the affection is met with in the puerperal state in which venous thrombosis is common. An analysis of the reported cases of sudden death from blocking of the pulmonary artery shows that, when careful search is made for the source of the embolus, failure to find it is very rare.

In some cases in which clots have been found in the pulmonary artery, and also in the veins of the extremities, the pulmonary symptoms have preceded the signs of venous obstruction elsewhere, and this fact has been adduced in support of the view that thrombosis, and not embolism, was the primary lesion in the pulmonary artery.

Evidence of this nature is not, however, conclusive, as signs of venous obstruction do not occur until obstruction is complete or nearly so. It must be remembered that a thrombus in a systemic vein is parietal before it becomes obliterative; and in the former condition, whilst the blood stream is still flowing over it, the clot or a portion of it may be detached and block a branch of the pulmonary artery. Subsequently a fresh thrombus may form at the original site, or, if any portion of the clot remain, additions may be made to it; and finally it may become obliterative, and only then give rise to signs of venous obstruction.

It is therefore quite possible that in cases of the kind under consideration the pulmonary symptoms may have really been due to embolism, although they preceded the signs of blocking of a distant vein. Whilst we are still of opinion that, in the great majority of cases in which the onset of the symptoms is sudden, embolism is the primary condition, we think that there is an increasing amount of evidence in favour of the view that spontaneous thrombosis occurs in the pulmonary artery with greater frequency than Cohnheim and most other writers are disposed to admit. Dr. Pitt,[1] indeed, considers that, so far from being very rare, it 'possibly occurs more frequently than in any other vein or artery in the body,' and that 'the clots in the pulmonary arteries, which some authors consider to be almost invariably embolic in origin, are, on a review of a number of cases, found to be more frequently due to thrombosis.'

Organisation of a thrombus.—In cases which recover, it is probable that the thrombus becomes organised in a similar manner to that observed to take place elsewhere in the body. The term 'absorption' is used by some writers in a manner which appears to imply that the clot may entirely disappear, apart from the ordinary process of 'organisation'; but that this should be so is *prima facie* improbable, and is, so far as the writer is aware, unsupported by pathological evidence.

The connective tissue, which in the process of organisation ultimately replaces the clot, may, it is true, by its contraction dilate the newly formed vessels within the thrombus, and, from this process of tunnelling, cavernous spaces may result, through which the blood ultimately finds its way, the lumen of the vessel being thus restored; but such a change is very rarely observed to occur, and in the great majority of cases a vessel once completely blocked remains obliterated, the process of organisation of the thrombus ending in the formation of a fibrous cord. Compensatory dilatation of neighbouring branches readily occurs in the pulmonary artery, but, owing to the absence of free anastomosis, this can only affect the terminal branches to a very slight degree.

Effects of thrombosis.—In some cases thrombosis is associated with hæmorrhage into the lung and the formation of an infarction, but it does not generally give rise to that lesion. Out of forty-six cases of the kind collected by Dr. G. H. Pitt from the post-mortem records of Guy's Hospital, a hæmorrhagic infarction was present in only eighteen.

Œdema, consolidation, and collapse are the pulmonary lesions which most commonly result from thrombosis of the pulmonary artery.

Symptoms.—Whether the sudden and severe symptoms elsewhere attributed to embolism ever arise from spontaneous thrombosis of the pulmonary artery must, we think, still be considered an open question.

During the formation of a thrombus in the pulmonary artery the character of the breathing and also the general condition may not be unlike that of a patient recovering from the most urgent symptoms of embolism when, indeed, secondary thrombi are not infrequently in process of formation; but the history of some cases—*e.g.* of advanced tuberculosis, malignant disease, or profound anæmia—clearly shows that extensive clotting may take place without the occurrence of any urgent symptoms, until the patient attempts some movement which throws a strain upon the heart. This is probably to be explained by the fact that in the condition of debility accompanying these diseases, the aeration of the blood can be adequately effected, whilst complete rest is maintained, although the sectional area of the pulmonary artery is considerably diminished.

When thrombosis occurs in pneumonia, as a rule no characteristic symptoms result; but Wilson Fox stated his belief that some cases which he observed of sudden death in that disease were due to this cause.

Case of thrombosis of the pulmonary artery.—The following case of thrombosis of the pulmonary artery, lately under the care of the writer in the Brompton Hospital, is an example of a rare condition, of which only a few examples are on record.

A woman, æt. 29, was admitted on September 28 and died on December 18, 1894. There was no history of rheumatism in early life. In 1889 she first noticed dyspnœa on exertion, and this symptom gradually increased. Two years later cyanosis appeared in the face. In 1891 she was admitted as a case of 'morbus cordis with cyanosis,' and again in March 1894 with a similar diagnosis. Since May 1894 the knees and ankles had been swollen and painful. There had been no hæmoptysis. Œdema had occasionally been present in the ankles.

The following notes were made on admission. 'The patient is extremely cyanosed, the face quite blue and the lips almost black. There is marked clubbing of the fingers, toes, and nose. The precordial area is prominent. The cardiac impulse is in the fifth interspace in the nipple line. The right cavities of the heart are enlarged. A very loud systolic murmur is audible at the apex, conducted outwards to the anterior axillary line and inwards to the left margin of the sternum. The second sound over the pulmonary artery is markedly accentuated.'

The degree of cyanosis varied from day to day, but the patient continued to suffer from severe dyspnœa and headache. On December 11 she became unconscious, and subsequently remained in a semi-comatose condition, with right hemiplegia and facial paralysis. Death was preceded by Cheyne-Stokes breathing. On post-mortem examination an abscess, containing fœtid pus, was found in the left inferior frontal convolution, extending deeply into the corpus striatum. The pulmonary orifice was of large size, and the valves were probably incompetent. The pulmonary artery presented a fusiform dilatation extending from the valves to the bifurcation of the vessel. This was lined with firmly adherent laminated clot, which extended to the point of division of each main branch of the artery. The lumen was occluded, but a probe could be passed between a softer part of the clot and the wall of the vessel. The walls of the artery were much thickened from the presence of numerous patches of atheroma, above the valves and throughout the branches distributed to each lung. The right auricle was greatly dilated, and the foramen ovale presented an opening, clearly at one period valvular, but which now admitted the tip of the little finger. The right ventricle was enormously hypertrophied, and the left was similarly affected but to a much less extent. The heart with the pericardium and vessels weighed 1 lb. 4 oz. The lungs were emphysematous and partially collapsed; they contained no infarctions.

In this case the atheroma was apparently the primary lesion. The long duration of the cyanosis and the extreme hypertrophy of the ventricle indicate that narrowing of the artery from thrombosis preceded the occurrence of dilatation of the vessel.

Diagnosis.—Such a case as that just described is very likely to be mistaken for one of congenital stenosis of the pulmonary orifice; but the age of the patient (29), and the fact that the symptoms had been noticed for only five years, negatived that diagnosis.

It will be apparent from the general description of the condition that in the majority of cases the symptoms are not sufficiently definite to permit of its recognition during life, although in a case of extreme anæmia or of malignant disease the presence of increasing dyspnœa and rapid action of the heart—for which no obvious cause can be discovered—associated possibly with signs of pulmonary œdema or consolidation, may suggest a correct diagnosis.

The **treatment** would not differ from that suitable in cases of embolism.

ATHEROMA AND SCLEROSIS OF THE PULMONARY ARTERY AND VEINS

The relative freedom from sclerotic changes observed in the pulmonary artery, as compared with the aorta and systemic arteries, is due to the much lower pressure to which, under normal conditions, its walls are subjected. If, however, the tension within be raised, atheroma occurs as readily there as elsewhere.

The most common conditions in which this lesion is found are mitral stenosis, mitral regurgitation and emphysema, all of which are accompanied by increase of pressure in the pulmonary vessels.

In a case of mitral stenosis we have observed an oval patch of atheroma in a large branch of the artery, nearly an inch in length and a quarter of an inch in depth on section. The change, as in a case previously narrated (*vide* p. 511), may extend to the smallest branches and even to the capillaries and pulmonary veins.

The larger trunks and secondary branches may undergo dilatation, and the valves may probably become incompetent, independently of organic change. We have, at any rate, observed a diastolic murmur over the site of the pulmonary valve in several cases of mitral stenosis in which there was no reason to suspect the existence of any lesion of the aortic valves. The murmur is variable; it may be present on one occasion and absent on another; its point of maximum intensity is in the second left interspace, and the line of conduction is down the left edge of the sternum. If the condition persists for any length of time, hypertrophy of the right ventricle occurs.

DILATATION OF THE PULMONARY VESSELS

The *pulmonary artery* may undergo dilatation as a result of extensive obstruction from thrombosis, or embolism followed by thrombosis, or when obstruction arises from the pressure of an intra-thoracic tumour or an aneurysm of the aorta, or from the presence of emphysema. A similar change has been observed in anæmia.

Dilatation of both vein and artery may arise from disease at the mitral orifice; and obstruction to the passage of blood into the left auricle, from the pressure of a tumour or aneurysm, may cause great dilatation of the veins.

The writer has observed cases of mitral disease in which a diastolic murmur was present over the pulmonary orifice, and was conducted downwards along the left border of the sternum, and in which all the usual signs of aortic regurgitation were absent. He is disposed to believe that, as a result of an associated dilatation of the pulmonary artery and right ventricle, the pulmonary valves, although free from disease, may become incompetent. It has been proved that a similar condition of the aorta and left ventricle may lead to aortic regurgitation in the absence of disease of the valves. Pulmonary diastolic murmurs of this nature are variable in character and may be observed to disappear under treatment.

J. K. F.

CHAPTER XLVI

ANEURYSM OF THE PULMONARY ARTERY

	PAGE
Etiology	514
Aneurysm in pulmonary tuberculosis	514
Number	515
Size	515
Site	515
Varieties	515
Clot in aneurysms	517
Rupture from disease of the vessel	518

Etiology.—1. Aneurysm of the pulmonary artery is most commonly met with in cases of *chronic pulmonary tuberculosis* in which cavities are present in the lungs. It also occurs, although rarely, in cases of *caseous tuberculosis* attended with rapid softening and excavation, and in cases of *bronchiectasis*.

2. A saccular or fusiform aneurysm of the main trunk may be produced by the bulging of the vessel wall over an area occupied by a patch of *atheroma*.

3. In cases of *ulcerative endocarditis* affecting the right side of the heart, if embolism of the pulmonary artery occurs, an aneurysm may form at the site of obstruction from increased pressure combined with inflammatory changes in the walls of the vessel, and whilst still unruptured such an aneurysm may contain laminated clot (Percy Kidd).

ANEURYSM IN PULMONARY TUBERCULOSIS

The most typical appearance is that of a rounded glistening gelatinous envelope projecting into a cavity, which in the majority of cases it does not nearly fill, but it may be almost equal in size to the cavity which contains it. In some cases the walls are thickened and opaque; in others this change may be limited to certain areas only.

The frequency of this lesion is now generally recognised, and at

DILATATION OF THE PULMONARY VESSELS

The *pulmonary artery* may undergo dilatation as a result of extensive obstruction from thrombosis, or embolism followed by thrombosis, or when obstruction arises from the pressure of an intra-thoracic tumour or an aneurysm of the aorta, or from the presence of emphysema. A similar change has been observed in anæmia.

Dilatation of both vein and artery may arise from disease at the mitral orifice; and obstruction to the passage of blood into the left auricle, from the pressure of a tumour or aneurysm, may cause great dilatation of the veins.

The writer has observed cases of mitral disease in which a diastolic murmur was present over the pulmonary orifice, and was conducted downwards along the left border of the sternum, and in which all the usual signs of aortic regurgitation were absent. He is disposed to believe that, as a result of an associated dilatation of the pulmonary artery and right ventricle, the pulmonary valves, although free from disease, may become incompetent. It has been proved that a similar condition of the aorta and left ventricle may lead to aortic regurgitation in the absence of disease of the valves. Pulmonary diastolic murmurs of this nature are variable in character and may be observed to disappear under treatment.

J. K. F.

CHAPTER XLVI

ANEURYSM OF THE PULMONARY ARTERY

	PAGE
Etiology	514
Aneurysm in pulmonary tuberculosis	514
Number	515
Size	515
Site	515
Varieties	515
Clot in aneurysms	517
Rupture from disease of the vessel	518

Etiology.—1. Aneurysm of the pulmonary artery is most commonly met with in cases of *chronic pulmonary tuberculosis* in which cavities are present in the lungs. It also occurs, although rarely, in cases of *caseous tuberculosis* attended with rapid softening and excavation, and in cases of *bronchiectasis*.

2. A saccular or fusiform aneurysm of the main trunk may be produced by the bulging of the vessel wall over an area occupied by a patch of *atheroma*.

3. In cases of *ulcerative endocarditis* affecting the right side of the heart, if embolism of the pulmonary artery occurs, an aneurysm may form at the site of obstruction from increased pressure combined with inflammatory changes in the walls of the vessel, and whilst still unruptured such an aneurysm may contain laminated clot (Percy Kidd).

ANEURYSM IN PULMONARY TUBERCULOSIS

The most typical appearance is that of a rounded glistening gelatinous envelope projecting into a cavity, which in the majority of cases it does not nearly fill, but it may be almost equal in size to the cavity which contains it. In some cases the walls are thickened and opaque; in others this change may be limited to certain areas only.

The frequency of this lesion is now generally recognised, and at the Brompton Hospital, in a fatal case of hæmoptysis, failure to find the ruptured sac is rare. It was discovered in every case of the kind examined during the year 1894.

Mode of formation.—Two causes are usually given for the dilatation of the vessel—viz. (1) lack of support to the wall, owing to the presence of the cavity; and (2) the extension of an inflammatory process to the coats of the artery. Of these, we are disposed to attach far more importance to the latter than to the former. Their relative influence was strikingly illustrated in a case of tubercular pyo-pneumothorax of long standing, recently under the care of the writer in the Middlesex Hospital. The patient died from recurrent hæmorrhage into the cavity of the pleura, after resection of portions of two ribs. The bleeding was proved, post mortem, to have been due to the rupture of an aneurysm the size of a pea, situated upon the internal mammary artery, and exactly resembling those commonly found upon the pulmonary artery in cases of tuberculosis of the lungs. In addition to the sac which had ruptured, there were upon the vessel four other aneurysms of a similar size. It was interesting to observe that, although the posterior aspect of the vessel was equally supported (or unsupported) throughout, aneurysms were only present where the inflammatory process in the pleura had extended to the wall of the artery, a clear proof that, in that case at any rate, the changes in the coats of the vessel, and not the lack of support, led to the formation of the aneurysms.

Number.—A single aneurysm is found in the majority of cases, but several may be present. A lung examined post mortem by Dr. Percy Kidd, and now in the museum at Brompton, contains twenty-two small aneurysms.

Size.—A common size is about that of a large green pea or a small cherry; they may, however, be miliary, or may attain to the size of an unshelled walnut or a small tangerine orange.

Site.—An aneurysm is generally found where an artery passes along the wall of a chronic cavity, leaving one aspect bare, and therefore liable to become involved in inflammatory changes taking place within the vomica. An aneurysm may also be formed in a small caseous patch which is undergoing softening, or a vessel may bulge into a dilated bronchial tube.

Varieties of aneurysm.—In the majority of cases they are rounded; but when a vessel crossing a cavity is dilated, a fusiform aneurysm results. Others more irregular in shape are sometimes met with.

a hemp seed, empty, and with thickened walls, arises from the same vessel as one of the preceding. The cavity is filled with blood clots.' In the other lung three aneurysms were found, and are thus described:—'Several small cavities in fibroid lung tissue, one of which contains a typical thin-walled aneurysm (globular) of about the size of a cherry stone, empty and unruptured; walls mostly thin, but in some places thickening has occurred. Just below this cavity is another, which is traversed by a fusiform aneurysm or aneurysmal dilatation, lying on one wall and exposed in most of its extent; walls thickened. In a neighbouring cavity there is another small aneurysm (miliary), unruptured and roughly cone-shaped, with a secondary bulging below its apex.'

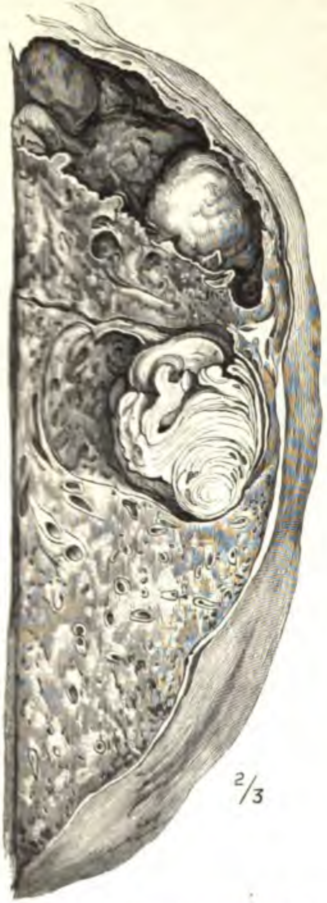


FIG. 125.—ANEURYSMS OF THE PULMONARY ARTERY IN CAVITIES IN THE LUNG

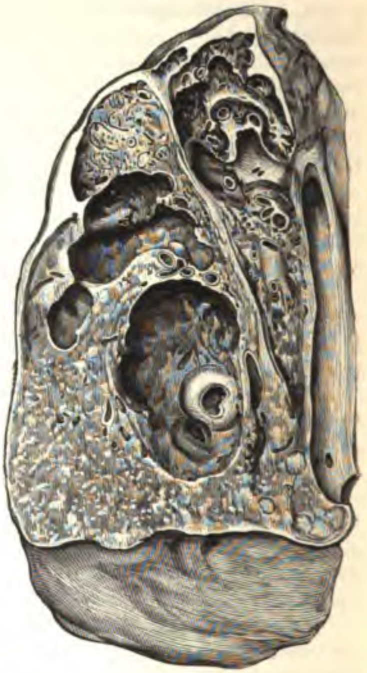


FIG. 126.—RUPTURED ANEURYSM OF THE PULMONARY ARTERY IN A CAVITY IN THE LUNG

A section of the lung from which fig. 125 is taken shows a ruptured aneurysm, of the size of a walnut, situated in a cavity in the upper lobe. In a cavity at the upper part of the lower lobe there is an aneurysm, of the size of a small marble, filled with soft clot, and surrounded by coagulated blood. The wall of the aneurysm can be seen as a curved whitish line crossing the mass of blood clot about its middle.

The coat is seen to be split, the two layers being separated by clot, the whole giving the impression that the blood first made its way between the coats of the wall and then burst through the outer wall.

In the specimen from which fig. 126 is taken a ruptured aneurysm is seen projecting into a cavity in the lower lobe of the lung. The aneurysm is attached to the wall of the cavity and a large vessel which can be traced up to the sac is seen to enter the aneurysm. The sac is uniformly thin and appears to be produced by a distension of the vessel. No laminated clot is present in the aneurysm.

Clot in aneurysms. It has been stated that laminated clot, similar to that found in aneurysms elsewhere, is never present in these aneurysms ; but this is not borne out by the examination of the specimens in the museum of the Brompton Hospital, several of which contain coagula of that character.

If rupture of the aneurysm has taken place the cavity in which it is situated is generally filled with clot, and it may be possible to distinguish that the portion of the clot contained within the sac is laminated, whilst that external to it has been produced by coagulation *en masse*, or a narrow slit may be found in the wall of the aneurysm, and the sac may be collapsed from the pressure of the extravasated blood. Rupture may be followed by cessation of the hæmorrhage, owing to closure of the opening in the aneurysm by the coagulation of the blood in the cavity, possibly aided by the formation of a thrombus in the vessel.

The condition found in a cavity in the upper lobe of the left lung of a patient who a short time previous to admission to the Brompton Hospital had suffered from a severe attack of hæmoptysis, is described as follows. 'The cavity is entirely filled by an aneurysm of the pulmonary artery, of the size of a filbert. The aneurysm has thin toughish walls and is distended by laminated thrombus. A large branch of the pulmonary artery leads into the obliterated aneurysm.'

The following is a description of a somewhat rare lesion present in a case of fatal hæmoptysis.

'In an irregular trabeculated cavity in the right upper lobe there is a cord, about half an inch in length, running from wall to wall, and constituted by a small artery and condensed lung tissue. It terminates in an aneurysm, of the size of a pea, attached to the cavity wall. The coats of the vessel are very thin, and have ruptured in a longitudinal direction close to the aneurysm.'

The conditions present in a case of repeated profuse hæmoptysis ultimately fatal are thus described.

'On incising the apex of the lower lobe a mass of about the size of a small orange, consisting of soft and laminated blood clot, escaped from a cavity. On one side of the latter there is a thin-walled aneurysm of the size of a walnut; a small perforation in this leads into the middle of the blood clot. A small decolorised clot is adherent to the inner wall of the aneurysm opposite the rupture. The blood clot in the neighbourhood of the opening is soft and resembles black currant jelly; that more distant is firmer and partially decolorised.'

RUPTURE FROM DISEASE OF THE VESSEL

The extension of either a tuberculous or a simple inflammatory process to the wall of the vessel commonly leads to the formation of a thrombus within, and the lumen is thus obliterated. If it were not for this, the majority of cases of pulmonary tuberculosis would be fatal from hæmoptysis at an early stage. The obliteration of the lumen of a vessel by the formation of a thrombus is more easily effected in a small artery than a large one. If a thrombus is not formed and softening of the lung tissue is in progress close by, the coats of the vessel ultimately become involved, and rupture takes place as soon as the wall is no longer able to withstand the pressure from within.

Rupture occurs in connection with tubercular lesions far more frequently than with any other form of disease, but it may be secondary to infiltration of the arterial walls in malignant disease or syphilis, to chronic congestion from valvular disease, to erosion from the pressure of an aneurysm upon the main trunk of the pulmonary artery, to the extension of a gangrenous process, and to chronic inflammatory changes of various kinds.

J. K. F.

CHAPTER XLVII

HÆMOPTYSIS

(HÆMORRHAGE FROM THE LUNGS)

	PAGE
Etiology	519
Vicarious hæmoptysis	520
Characters of expectorated blood	520
Morbid anatomy	521
Symptoms	521
Illustrative case	522
Examination of the chest	523
Diagnosis	523
Spurious hæmoptysis	524
Prognosis	525
Treatment	525

HÆMOPTYSIS occurs when blood which has escaped into the air passages is expectorated.

If the space into which the blood is poured out is no longer in direct communication with a bronchus, extravasation of blood may occur without hæmoptysis. For example, a pulmonary aneurysm may rupture into a cavity and none of the blood may be expectorated; this, however, rarely occurs.

Etiology.—The most common causes of serious hæmoptysis are (*a*) the rupture of an aneurysm on a branch of the pulmonary artery, and (*b*) the rupture of a branch of that vessel from the presence of tubercular or other disease in its walls. These conditions have already been described (*vide* p. 515 *et seq.*).

(*c*) Hæmoptysis may also occur from active or passive *hyperæmia* either of the bronchial or pulmonary capillaries. The quantity of blood thus lost is usually small in comparison with that which results from either of the lesions previously mentioned, but we have known profuse and fatal hæmoptysis to occur in a case of miliary tuberculosis in which, on post-mortem examination, no lesion was found in any vessel of considerable size, the changes present clearly proving that the hæmorrhage had occurred from a great number of small vessels.

Hyperæmia followed by capillary hæmorrhage may occur in the early stage of tuberculosis, in bronchitis, and in any inflammatory

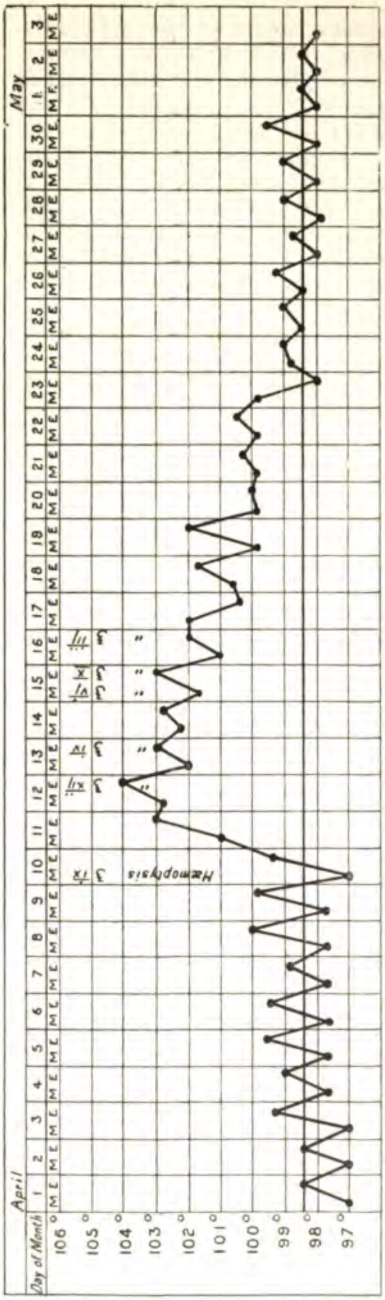


FIG. 127.—INITIAL LOWERING OF TEMPERATURE AND SUBSEQUENT PYREXIA ACCOMPANYING AN ATTACK OF HÆMOPTYSIS

is equally true of cases of primary hæmoptysis and of those in which a previous severe attack has occurred. Tubercular patients are proverbially hopeful, but they are almost invariably alarmed by hæmorrhage.

A primary attack in a young subject previously in good health may be recovered from in a few days, or the bleeding may recur after a short interval. Profuse hæmoptysis in an advanced stage of the disease may be repeated at intervals of a day or two for a long period and may finally end fatally. Such a condition always suggests that an aneurysm of the pulmonary artery has ruptured and is leaking through blood clot in a cavity. The following is an abstract of the notes of a case of this nature.

A male, æt. 26, suffering from pulmonary tuberculosis, was admitted to the Brompton Hospital on August 30 and died November 16, 1886. Fifteen weeks previously he had an attack of hæmoptysis. Two weeks later he was said to have brought up half a gallon of blood.

October 29.—Hæmoptysis 11 oz.

October 30.—Hæmoptysis continues. Temp. 103·4°.

October 31.—Hæmoptysis 2 oz.

November 9.—Hæmoptysis 7 oz. Respirations 60.

November 10.—Hæmoptysis 12 oz.

Pyrexia persistent, with moderate remissions. During the night he had another attack of bleeding, which was immediately fatal. *Post mortem.*—The right upper lobe contained two trabeculated cavities. In one of these there was a large rounded blood clot partly covered by a thin capsule, which had formed a portion of the wall of a freely ruptured aneurysm. A sinuous channel through the clot was continuous with a large branch of the pulmonary artery. There was also an unruptured miliary aneurysm upon a trabecula situated in a recess of the same cavity.

Examination of the chest.—During the continuance of the bleeding, and for some time afterwards, the patient should not be moved for the purpose of examination, or asked to take a deep breath, cough, or speak, and the chest should not be percussed. Inspection of the chest and auscultation over the upper lobes in front during quiet breathing can do no harm, and may give indications as to which lung is affected. The heart sounds should also be auscultated in order to exclude the presence of valvular disease. If tubercular disease of the lungs was known to exist previously to the attack of hæmoptysis, examination may prove the presence of excavation; and it is well, if possible, to be aware of this, as it materially influences the immediate prognosis.

Diagnosis.—This presents but little difficulty if one is at hand during the attack, but it is often necessary to determine from the history given by the patient, whether blood has been vomited or has come from the respiratory passages, and with hospital patients this is not always easy. The point can generally be decided by the answers to the following questions:

1. Was the attack preceded by pulmonary symptoms (cough &c.) or gastric symptoms (pain after food, &c.)?
2. Was the expectoration bloodstreaked on the day following the attack?
3. Did the blood come up all at once or in successive mouthfuls?
4. Was faintness present before the blood appeared?

It is almost useless to ask a stupid patient whether he was coughing at the time or vomiting.

In hæmatemesis the blood is acid, of a darker colour, grumous appearance, and may be mixed with food. The writer once saw a case which had been diagnosed as 'phthisis, gastric ulcer, hæmorrhage from bowel.' It is almost needless to say that it proved to be one of hæmoptysis, in which some of the blood had been swallowed and subsequently vomited, and the remainder passed by the bowel.

In all cases of primary hæmoptysis the expectorated blood should be examined for tubercle bacilli; they may be found, even when the patient was thought to be in good health up to the time of the attack. It is (apparently) extremely common for patients who have fulfilled. The statement above made as to the occasional absence of physical signs after hæmoptysis really due to tubercular disease, applies rather to fairly muscular adult males than to anæmic young females.

We can, however, recall a case in which the diagnosis presented great difficulty. The patient was a well-built man, about forty years of age, addicted to alcohol and morphia. Large quantities of blood were brought up at long intervals, but there was no cough, and no physical signs developed in the lungs. The blood was alkaline in reaction, of a bright red colour, mixed with air, but did not coagulate in lumps in the vessel, and presented a more uniform appearance throughout than is usually observed in hæmorrhage from the lungs. There was no evidence of disease within the mouth, larynx, trachea, or lungs. Various opinions had been given as to the source of the bleeding: the writer inclined to the view that it came from the pharynx or the upper part of the œsophagus.

Prognosis.—Hæmoptysis occurring in the course of pulmonary tuberculosis should never be made light of. It is true that in the early stage of infiltration an attack is very rarely fatal, although we have known death occur in a young child from the rupture of an aneurysm of the pulmonary artery, when the total area of the lung affected could be covered by the tip of one finger; and it must be remembered that in cases of tuberculosis the extent of the disease in the lungs is almost always much greater than is indicated by the physical signs. Profuse and repeated hæmorrhage in advanced tubercular disease is a very grave condition, as it points strongly to the rupture of an aneurysm; but fortunately the repair of such a lesion is possible.

A small bleeding in such a case may, however, be the first sign that an aneurysm is leaking, and as it is impossible to be certain of the exact condition, a guarded prognosis should always be given.

In a case of aneurysm of the thoracic aorta, even though the quantity of blood expectorated is small, as at first it may be, the possibility of the rupture of the sac having occurred must be remembered in forming a prognosis.

In mitral disease hæmoptysis, secondary to infarction, usually points to the presence of extreme pulmonary congestion, which is often accompanied by failure of the right side of the heart, and life may in such cases be indefinitely prolonged after the occurrence of hæmoptysis. If it is secondary to ulcerative endocarditis of the tricuspid or pulmonary valves, the outlook is extremely unfavourable. This is a grave condition, but in cases of mitral stenosis accompanied by hæmoptysis, we have known recovery to follow when the state of the patient appeared to be almost hopeless.

Treatment.—Capillary hæmorrhage secondary to hyperæmia is rarely attended by profuse bleeding, and the discharge of blood tends by itself to relieve the condition.

When there is reason to suspect that the hæmoptysis depends upon the erosion of a large vessel or the rupture of an aneurysm of the pulmonary artery, the indications for treatment are to favour the formation of a thrombus, which is the natural method of repair of such a lesion. This is to be effected by (1) diminishing the movement of the lungs, (2) lowering the blood pressure generally, and in the pulmonary artery particularly, and (3) slowing the circulation.

Rest fulfils all these indications; therefore on the appearance of merely blood-stained sputum the patient should immediately go to bed, and the necessity for so doing is more obvious if an appreciable quantity of blood has been expectorated. By a timely resort to bed it is extremely probable that a more serious hæmorrhage may be averted. The room should be cool and the clothing light. Talking and movement should be avoided. Cough may be checked by sucking small pieces of ice, or, if severe, by the administration of a sedative linctus. No hot food of any kind should be given, and stimulants are absolutely to be avoided.

The following 'diet for hæmoptysis' in use at the Brompton Hospital indicates the nature of the food suitable under such circumstances.

Milk	$1\frac{1}{2}$ pint
Meat Essence . . .	2 oz.
Bread	4 oz.
Butter	$\frac{1}{4}$ oz.

The bread and butter to be given as small thin sandwiches.

The bowels should be freely opened by the aid of saline purgatives, a draught containing a drachm of sulphate of magnesia and twenty grains of sulphate of soda being given every four hours. The effect is to lower the general blood pressure, and consequently that within the pulmonary artery, by the withdrawal of fluid in the stools, and by the determination of blood to the intestinal veins.

In cases of a moderately severe type, the writer regards free purgation as an essential, and probably the most effective measure which can be employed in the treatment of hæmoptysis occurring in tubercular disease of the lungs.

If the hæmorrhage is profuse a hypodermic injection of morphia (gr. $\frac{1}{4}$–$\frac{1}{3}$) should be given at once. Under its influence mental distress is relieved, and the action of the heart, which may have been excited, quiets down.

It may be necessary to repeat the injection of morphia, and in severe cases to keep the patient to a certain degree under the influence of the drug for some days.

It is very seldom that the use of morphia is contra-indicated by the fear of increasing the tendency to asphyxia, threatened by the accumulation of blood in the bronchi, but this is a danger which must be borne in mind. When the blood is in the mouth the patient should be encouraged to expectorate it freely.

Drugs which directly affect the blood pressure, such as nitrite of amyl and nitroglycerine, are inadmissible in the treatment of hæmoptysis, owing to their stimulant effect upon the heart. Aconite and tartar emetic are useful from their action as vascular depressants, but in cases of tuberculosis they should be used with much caution and with due regard to the strength of the patient, and when administered should never be given in sufficient doses to produce vomiting.

Whilst the blood is coming up freely the patient is fully occupied in getting rid of it, and cannot retain anything in the mouth, but during the intervals he may be allowed to suck small pieces of ice. It is possibly true that this can have no direct effect upon the bleeding vessel, but it is a measure which is free from possibility of harm, and by producing in the mind of the patient a feeling that something is being done for his relief, tends to diminish nervous excitement.

The application of an icebag to the chest is not a mode of treatment from which, in the writer's experience, any benefit has resulted. In hæmoptysis from the rupture of an aneurysm—and most of the serious attacks are due to that cause—it is hardly possible that it could be of any service.

There are few points in therapeutics upon which more divergent opinions exist than as to the value of astringent remedies in the treatment of hæmoptysis. Considering the long period of time during which they have been employed, this fact by itself tells somewhat against their utility. As in the great majority of cases of hæmoptysis with a limited area of disease the bleeding ceases spontaneously, provided the patient keeps absolutely quiet and observes the ordinary rules which prudence suggests, it is obvious that under such circumstances any remedy which is given sufficiently often may obtain an undeserved reputation. Until it was known that pneumonia tends to recovery after a duration of about seven days, and that the febrile period in an ordinary attack of influenza generally lasts about three or four days, a variety of drugs were credited with the cure of those diseases. The fact that astringents are on *à priori* grounds most likely to be of service in capillary hæmorrhage, in which spontaneous arrest occurs most readily, still further increases the difficulty in coming to a conclusion. As already stated, serious hæmorrhage is almost invariably due either to the erosion of a vessel from the extension of tubercular disease to its walls, or to the rupture of an aneurysm of the pulmonary artery. In such conditions it is, to say the least, extremely improbable that any contractile elements remain either in vessels thus diseased or in such as traverse the indurated walls of chronic cavities. The only effects which are certain to follow the use of such drugs as ergot, ergotine, gallic acid, hamamelis, acetate of lead, &c., are the contraction of healthy vessels, a rise in the general blood pressure, and constipation. Whether it is wise to risk so much in order to obtain such an uncertain result as the possible contraction of the ruptured vessel is very doubtful. Since the writer ceased, many years ago, to use these remedies he has not observed that attacks of hæmoptysis not so treated are more prolonged than they were formerly.

In cases of valvular disease, and in others in which the action of the heart remains excited after the mental disturbance which accompanies the first appearance of the bleeding has passed away, digitalis may be given with advantage. The good effects which follow a slowing of the cardiac action far outweigh the disadvantages attendant upon a moderate increase in the arterial tension.

Turpentine, although an astringent, also acts as a vascular depressant, and may be valuable for the latter reason.

After the sputum has been free from staining for a few days, the patient may be allowed to get up ; but he should be carefully watched for a considerable period, and should abstain from any violent exercise for some months, and avoid alcohol and tobacco. We have seen several cases of primary hæmoptysis in young men whose lungs were doubtless already diseased, although they were not aware of the fact, in whom the attack occurred during the night after violent action of the heart induced by excessive smoking. In a case of primary hæmoptysis a change of climate is indicated, so soon as the patient has recovered sufficiently to bear a journey.

Spurious hæmoptysis in anæmic girls should be treated by attention to the menstrual functions, the administration of iron in the form of Blaud's pills, and the use of astringent applications to the gums and, if necessary, to the naso-pharynx. The frequent association of constipation and anæmia renders attention to the bowels especially necessary in these cases. Saline purgatives are usually the most suitable remedies for the constipation.

The treatment of hæmoptysis accompanying the numerous affections named at the commencement of this chapter should, speaking generally, be conducted on the lines here recommended, but with certain modifications which hardly need to be considered in detail. When, for example, the amount of blood expectorated is very slight, and from the nature of the disease the presence of an aneurysm of the pulmonary artery can be excluded, it may not be necessary to adopt quite such a rigid treatment as regards rest, diet, &c. as is advisable in cases of pulmonary tuberculosis. We may, however, repeat that even in these cases, if the medical attendant is disposed to regard the symptom as of little importance, he will rarely find that his views are shared by the patient.

J. K. F.

CHAPTER XLVIII

PULMONARY COMPLICATIONS OF SOME ACUTE SPECIFIC DISEASES

	PAGE
Influenza	529
Morbid anatomy	529
Tracheo-bronchitis	529
Broncho-pneumonia	530
Pneumonia	530
Symptoms	531
Physical signs	532
Treatment	532
Whooping cough	533
Morbid anatomy	533
Glanders (Farcy)	534

INFLUENZA

THE bronchial and pulmonary lesions of influenza are of special importance owing to their marked influence upon the mortality from the disease. Except in cases fatal from cardiac failure, death rarely occurs unless some affection of the respiratory organs is present.

This may take the form either of an acute tracheo-bronchitis, a diffuse broncho-pneumonia, or a lobar pneumonia.

Morbid anatomy.—*Tracheo-bronchitis.*—The lining membrane of the trachea and bronchi is deeply congested and acutely inflamed, and the tubes contain viscid and possibly blood-stained mucus, which may increase in quantity as the smaller divisions are reached. These often show intense reddening of the mucous membrane, which may present a villous appearance. At a later stage the bronchial wall may be found softened and the tubes may have undergone some degree of dilatation and be filled with thick grey muco-purulent secretion. The lungs are often much distended after death, doubtless as a result of the frequent cough. They are described as being peculiarly dry when the case has been fatal at becomes bathed in cold perspiration, the strength gradually fails, and death follows.

Physical signs.—When the disease is limited to the trachea and larger bronchi, the respiratory sounds may be but little altered, or perhaps somewhat harsh in front, but feeble at the bases.

Sibilant rhonchi may be present, but the ordinary signs of bronchitis are not a marked feature of influenza. When the inflammation has extended to the finer tubes, a peculiar sharp high-pitched râle of a somewhat 'hissing' quality may be audible, especially at the bases of the lungs.

By some writers these sounds are described as 'sharp and sticky.' One became so familiar with them during the late epidemics that it was almost safe from their presence alone to express a confident opinion that the case was one of influenza. However, in a case which proved to be one of acute tuberculosis of a most unusual character seen by the writer, precisely similar râles were heard. Fortunately, the sputum was at once examined for tubercle bacilli, and their discovery rendered the diagnosis clear.

When broncho-pneumonia is present, there may be no obvious impairment of the percussion note even at the bases, and no signs of complete consolidation of any definite area; but crepitation will be heard over the lower lobes, and this sign is usually most marked. The breath sounds are generally feeble, but if the foci in a given area are so numerous as to produce almost complete consolidation, tubular breathing may be audible, and the percussion note will be dull. This condition is, however, the exception rather than the rule.

The physical signs which attend a true pneumonia do not differ from those described elsewhere (p. 213).

Treatment.—Early resort to bed is advisable in all cases, and it is well for the patient to remain there until all adventitious sounds have disappeared from the lungs, and the temperature has been normal for at least four days.

There is a general agreement that the pulmonary complications of influenza require a stimulant mode of treatment, and that depressing remedies of all kinds are to be avoided.

The extreme viscidity of the bronchial secretion suggests the use of warm stimulant alkaline fluids, as recommended in the chapter on Bronchitis (p. 108). By diminishing this viscidity they aid expectoration, and thus give far more relief to the cough than is obtained from the administration of sedative remedies.

If there is high fever, sponging the body with tepid water often affords relief, or hydrobromate of quinine (gr. 4) combined with a very small dose of phenacetin (gr. j) may be given hourly for six or eight doses (Yeo).

Mustard leaves or hot linseed meal poultices are the best local applications.

The microscopical changes are those which commonly attend acute inflammation of the bronchi, broncho-pneumonia and pneumonia, and, in addition, the broncho-pneumonic areas and the peribronchial lymphatics contain continuous masses of minute non-motile bacilli (Pfeiffer's influenza bacillus). Similar organisms are found in the sputa, either singly, or in small groups, or in larger masses. In some portions of the sputa they occur almost in the form of a pure culture (Klein). During the acute stages they are very numerous in the sputa, but are less so as the disease subsides.

Symptoms.—The bronchial and pulmonary symptoms in succeeding epidemics of influenza, and in different individuals during the same epidemic, present great variety. At the outset of the series of epidemics from which this country has recently suffered the coryzal mode of invasion was rare, but of later years it has appeared to be more common.

In a large number of cases occurring in the recent epidemics the pulmonary complications appeared after the initial pyrexia had subsided, and were attributed to the patient having 'caught cold' from some imprudence. They are, however, far more often a manifestation of the continuance of the disease than of an ordinary catarrhal inflammation of the air passages.

The most marked symptom of the tracheo-bronchial lesion is a very distressing, short, hard, frequently repeated cough. It is generally dry, but may be accompanied by a scanty expectoration of glairy mucus.

Cough may appear at the onset of the disease, or not until a few days later, when the lungs have become affected. It is of a very persistent character and difficult to relieve. Hoarseness may also be present. The breathing is almost invariably quickened, and if dyspnœa is present it may be out of proportion to the physical signs indicating obstruction of the air passages. The expectoration, at first glutinous and scanty, subsequently becomes more copious and is expelled with less difficulty. When the muco-purulent stage has been reached, the sputa may be excessive in quantity.

It is not easy to determine when capillary bronchitis ends and broncho-pneumonia begins, as in the majority of cases the latter is but an extension of the same morbid process. If the alveoli become involved the pyrexia continues, the sputum may increase in quantity, but it is still viscid and difficult to expel. The cough continues of the same character. The respirations increase in frequency and the face appears either flushed or congested, and the signs of prostration become more marked.

If the disease pursues an unfavourable course, the area in which the physical signs are present gradually extends as successive parts of the lung become involved, dyspnœa is followed by orthopnœa, the pulse becomes more rapid, feeble and intermittent, and the signs of impeded pulmonary circulation and of over-distension of the right side of the heart are more obvious. Delirium appears, the skin (*d*) True *emphysema*, as stated in the chapter on that subject, is less common than a general over-distension of the lungs.

(*e*) The occurrence of subpleural and mediastinal emphysema and pneumothorax is described in Chapter XIII. (p. 180).

Pleurisy often follows from the extension of pneumonia to the surface of the lung.

The **morbid anatomy** and the **treatment** of the various affections here enumerated are fully dealt with under the appropriate headings.

GLANDERS (FARCY)

Glanders is a disease due to the presence of a specific organism, the *bacillus mallei*, and is communicated to man from the horse, donkey, or mule. There is both an acute and a chronic form of the affection, the former being much more common in the human subject than the latter; but even the acute form is a very rare disease.

The pulmonary complications of the disease are almost invariably secondary to specific lesions of the mucous membrane of the nose of the characteristic granulomatous type, and softening of the nodules is followed by the appearance of ulcers; the destructive lesions may extend to the septum, turbinated bones, and palate. If, as is sometimes the case, the lesions are confined to the upper part of the nose, no ulcers may be visible. It should not be forgotten that when the nasal symptoms are not much in evidence, the diagnosis is often extremely difficult, and that cases of acute glanders are not unfrequently mistaken for rheumatism or pyæmia, and in such cases it is not unlikely that the pulmonary symptoms may attract special attention.

The disease subsequently extends to the bronchi, and excites similar inflammatory changes in the mucous membrane, attended by severe cough and profuse purulent expectoration. The characteristic lesion of glanders affecting the lungs consists of the presence of firm hard nodules—some immediately beneath the pleura, others more deeply situated. They vary in size from that of points just visible to the naked eye to that of walnuts, the most common size being that of peas. One or two only may be present, or as many as several hundreds may be found in each lung, but they are not so numerous as are commonly the nodules of tubercle. On section they are of a pearly grey colour, some with a peculiar yellowish white centre and are surrounded by a dark hæmorrhage-like zone (M'Fadyean). Around these areas more or less extensive pneumonia is observed, and sometimes dark hæmorrhagic areas may be seen in which the grey nodules are scattered. The process in the lungs is more acute in man than in the horse, and there is more congestion round the pneumonic areas, which moreover have a much greater tendency than in the horse to undergo softening. The nodules when firm may be sometimes shelled out of the surrounding lung tissue, and in the chronic form of the disease they may become hard and occasionally calcified. In the latter cases it appears that the bronchial and mediastinal glands may be similarly affected.

The physical signs of the bronchial lesions are rhonchi, which may be audible over the greater part of both lungs. Pneumonia and necrosis are attended by the ordinary signs of consolidation and breaking down of the lung.

J. K. F.

CHAPTER XLIX

INJURIES OF THE PLEURA

	PAGE
Contusion	536
Laceration	536
Punctured and incised wounds	536
Septic infection of the pleura	538
Hæmothorax from wound of the pleura	538
Foreign bodies in the pleura	538
Traumatic diaphragmatic hernia and wounds of the diaphragm	539
Congenital diaphragmatic hernia	541
Acquired diaphragmatic hernia	542

INJURIES of the pleura are so frequently associated with injuries of the ribs that it is almost difficult to think of them in any other connection. There are, however, several other conditions that merit our attention.

Contusion.—So many cases have been recorded where pleurisy, with or without effusion, and even empyema have followed blows upon the chest without obvious injury to bone that it behoves us to keep an open mind, at all events, as to the possibility of this sequence of events. Subpleural hæmorrhage, either beneath the visceral or parietal part, may certainly be caused in this way, especially in the flexible chests of the young. Probably a certain amount of hæmothorax may arise in the same manner, but this generally presupposes a laceration of the pleura.

Laceration.—Extensive laceration may occur without injury to bone, as in the passing of a cart-wheel over the chest of a child; but such an accident will almost certainly lead to serious injury of viscera, and therefore does not merit separate consideration.

Punctured and incised wounds.—Different opinions have been expressed as to the immediate effect upon the lung of a punctured or incised wound of the pleura. There is no doubt that a simple puncture or incised wound does not necessarily lead to collapse of the lung; and it is also certain that in some morbid conditions, as, for example, in some cases of empyema, when the pleura is opened freely, the lung promptly fills up the cavity previously occupied by the pus. But it would be a great mistake to argue from these facts that a free incision may be made into the pleura without causing collapse of the lung, or that the lung will always come out to the ribs when an empyema is freely opened. The two conditions are obviously totally different, and, as the latter part of the subject is fully dealt with in the chapter on Empyema, we will now only direct our attention to the question of punctured and incised wounds.

The subject is not really so complicated as some writers would have us believe. When a simple incised wound has been made in an intercostal space without disturbing the lung, the visceral and parietal layers of the pleura are kept in contact with one another precisely as a boy's 'sucker,' when moist, is kept adherent to the paving stone, or as a wet cover glass is held down to the microscope-slide. But if the finger be pushed into the wound, or if a second incision be made at the front of the chest, the same result is brought about as if a needle be pushed in at one spot between the cover glass and the slide, or as if a small amount of air were to enter between the sucker and the paving stone: the lung recedes and assumes its normal collapsed size—not of course quite that of the perfectly normal dead lung as we see it in the post-mortem room, because it now contains its full supply of blood; but, on the other hand, it is, if healthy, much smaller than many of the lungs we see distended by congestion or œdema or solidified by inflammation. It is easy to convince oneself of this by performing the experiment upon an anæsthetised guinea-pig, when the observer will also have the opportunity of convincing himself that the movement of inspiration has no effect in producing even the slightest expansion of the lung. It is indeed self-evident that such must be the case. Efforts at expiration with a closed glottis can no doubt drive some air from the opposite side into the collapsed lung, but that is all that any movement of respiration can possibly do. These facts are well known to the practical surgeon. He knows that if he incises a healthy pleura and puts his finger in, the lung always recedes to a greater or less extent. Most of us have met with this accident in searching for pulmonary abscesses, and many in the course of operations for opening hepatic abscesses through one of the lower intercostal spaces. The surgeon also knows that there are certain situations, such as the apex of the pleura, where even a simple puncture leads to a pneumothorax; more than once or twice a slight sucking in of air has followed a puncture of the pleura in the posterior triangle during such operations as the removal of deep glands or the cutting away of an accessory cervical rib. The rapid recovery from such a condition, if the wound be closed, depends upon the absorption of the air by the pleura, and is precisely analogous to the cure of a pneumothorax following fractured ribs.

The practical outcome of what has been said is that if a penetrating wound of the pleura has been made with a surgically clean instrument the right treatment is to close it without delay, in confidence that the rest of the cure may be left to nature.

But unfortunately other things besides air may make their entrance at the time of the injury—viz. (1) septic organisms; (2) blood; (3) foreign bodies—and these require separate consideration.

Septic infection of the pleura.—If the wound have been made with a dirty instrument, a septic pleurisy and empyema will almost certainly result. But seeing that it is impossible in many cases to ascertain this at the time of the injury, the correct practice generally is to close the wound and watch the patient for a few hours. The signs of septic infection will soon manifest themselves, and, if they do so, no time should be lost in freely opening up the wound, probably aiding this by excising a portion of a rib. A large tube should be inserted, and in fact the case should be treated like any other septic empyema. It is well under these circumstances to give a very guarded prognosis. My experience is that while in a certain number of cases of this sort a rapid cure is obtained, there are not a few in which the lung remains collapsed and the condition becomes chronic. Sometimes a cure may be then obtained by means of a formidable Estlander's or Schede's operation; sometimes the patient is, I believe, fortunate enough to obtain a respectable amount of health by the closure of the external wound over a permanent pneumothorax, the presence of which is then probably not even suspected and scarcely diagnosable.

Hæmothorax from wound of the pleura.—If an intercostal artery or the internal mammary be wounded by the instrument which has entered the thorax, a large amount of blood may collect in the pleura in a short space of time. The patient will exhibit all the signs of hæmorrhage, and the surgeon must seek for the bleeding point and tie it without delay, or the patient may die either from hæmorrhage or from asphyxia. This may very likely necessitate the removal of a piece of the rib or rib cartilage behind which the vessel is situated. It will probably be also advisable to open the pleura to extract the clots, and afterwards to close the opening.

If, however, the amount of blood be small and there be no reason to suppose that the vessel from which it has come is continuing to bleed, the case may be left alone and the blood will be absorbed. It will not be forgotten that a small amount of blood, if it become clotted, may give rise to an amount of dulness out of proportion to its quantity, and that it is not absorbed so quickly as air or serum. This condition is, of course, frequently met with as a complication of fractured ribs. Blood, however, usually remains for long uncoagulated in the pleura.

Foreign bodies in the pleura.—The instrument causing a wound may break, or the foreign body may be a shot or bullet or other missile, or even a drainage tube insecurely held in position. It is needless to say that if it be quite certain that such a foreign body is lying free in the pleura, it should not be allowed to remain there; and that, if the pleura be healthy, it will gravitate to the most dependent part. Thus if it be not found at once at the seat of injury, the foreign body will probably be met with at the lowest part of the pleura behind. If, therefore, it cannot be extracted by means of long forceps introduced through the original wound another incision should be made behind, just above the lowest limit of the pleura. The exact extent of the pleura in any particular case can usually be ascertained by means of a long probe introduced through the opening already present.

Wounds of diaphragm and traumatic diaphragmatic hernia.—Wounds of the diaphragm, whether made by cutting instrument or gunshot, or caused by forcible compression of the chest, may be followed by serious intrapleural hæmorrhage, and if there be an external wound by external hæmorrhage which must be treated upon general principles.

A much more serious consequence, however, is the formation of a diaphragmatic hernia. There appears to be no tendency, if the diaphragm be wounded, for the lungs to make their way into the cavity of the peritoneum, and this might, *à priori*, be anticipated because the natural tendency of the lung is to collapse, and the hollow viscera on the lower surface of the muscle are more or less analogous to the external air and pass through the rent either into the pleura or the pericardium. It is conceivable that if a sudden cough occurred at the moment of the infliction of the wound, the lung might be forced through the opening, as happens in rare cases where the lung protrudes through a wound made in an intercostal space; but it is most unlikely that it should be nipped in an opening in the diaphragm and prevented from returning. Occasionally the intestines have not only entered the pleural cavity, but have actually protruded through an external wound of the chest wall.

Traumatic diaphragmatic hernia appears to be more common on the left side than the right, for which two explanations have been given: first that, from the nature of the case the wound—being most usually directed by some right-handed person against the presumably most vital part of his opponent—is most likely to reach the left side of the chest; and, secondly, that the presence of the liver on the right side prevents the passage of the hollow viscera through an opening which on the left side would supply an easy route for their ascent.

The opening may vary from an extensive rent of the diaphragm, allowing of the immediate passage of large quantities of the abdominal viscera into the chest, to a minute hole which perhaps admits only a small part of the stomach or intestine. In the latter circumstance, or where the opening is of medium size, it is possible that, as time goes on, more and more of the abdominal contents may be drawn or pushed upwards, and the symptoms, slight or equivocal at first, may ultimately become serious and unmistakable.

Most of these traumatic herniæ do not possess a sac; but sometimes, if the injury consists of a rupture of muscular fibres, there may be a distinct sac consisting of either peritoneum or pleura, or of both these membranes.

The *symptoms* depend partly upon the injury of the diaphragm itself and partly on the interference with the functions of the abdominal and thoracic viscera.

The symptoms attributable to injury of the diaphragm, which would consist in modifications in the respiration, are not to be distinguished from those that depend upon interference with the functions of the thoracic viscera. But if the hernia is small the dyspnœa, which is almost always present, must probably be attributed to interference with the movements of the diaphragm. If, however, a large amount of the abdominal contents have entered the chest, not only will the lung be collapsed and perhaps pressed upon, but the heart will be dislocated to the opposite side. We have thus a complication of causes leading to the dyspnœa which, as has been said, is usually one of the most prominent symptoms.

The abdominal symptoms will depend upon the particular viscera which have been displaced. Of these the stomach is the most frequently met with; but the spleen, the small intestine, the colon, the liver, and the mesentery, and more rarely the duodenum, the pancreas, and the cæcum have all been found in diaphragmatic herniæ. It results from this that vomiting is one of the most constant symptoms. But if the patient should not succumb to the injury, others attributable to the inclusion of the other organs may in time manifest themselves, such for example as constipation, colic, dragging pains at the umbilicus and the symptoms we are accustomed to associate with the presence of hernia elsewhere. The patient will probably emaciate, and it is possible that the ordinary symptoms of strangulation may supervene. Peritonitis and pleurisy are also very likely to occur. Should the stomach or intestine be wounded, the presence of their contents either in the pleura or at the external wound will practically settle the question of the nature of the injury.

The *physical signs* will probably be modified by the presence of hæmothorax, and possibly by that of blood in the abdomen. The most striking physical sign, however, is likely to be the presence of a tympanitic area at the lower part of the chest, over which no breath sound is to be heard; but possibly the gurgling sounds caused by the movements of the stomach or intestines may be detected. Displacement of the heart will materially aid the diagnosis.

The *prognosis* of these cases is bad; most of them ultimately end fatally, either rapidly from dyspnœa, or later by interference with the digestive functions. But it is not hopeless, for a certain number who have survived the immediate effects of the injury have lived for very considerable periods, and some have suffered comparatively little inconvenience, the actual condition of things being scarcely suspected or only surmised.

The *treatment* must be considered as still *sub judice*. A sufficient number of cases has not yet occurred since the advent of what may be called modern surgery. They used to be considered quite beyond the reach of surgical aid. Then it was suggested that an attempt might be made to reduce the hernia by introducing a small hand into the rectum and pulling the viscera back into their natural position. This was not a practical suggestion, and was never favourably received by the profession. Next it was suggested that an abdominal incision should be made, and the reduction performed by traction, after, if necessary, enlarging the opening. This would probably be a very difficult procedure, and it seems very unlikely that, even if it were performed, anything could be done in the way of remedying the defect in the diaphragm by suture.

The most rational suggestion has been made since the comparatively recent improvements in thoracic surgery—namely, that a free opening should be made into the chest by removing portions of a sufficient number of ribs, and that after the viscera have been pushed back through the opening, it should be closed by sutures. Speaking without any personal experience, I must say that this seems not only the most likely method of affording relief, but also rational, and one that ought to be put in practice. The chief objection to it is that it adds pneumothorax to the previously existing dangers of the patient.

Congenital diaphragmatic hernia.—It is well before leaving this subject to say a few words about congenital and acquired diaphragmatic hernia.

Congenital diaphragmatic hernia depends upon some congenital defect of the diaphragm, which may amount to a small opening only, or may involve the absence of one-half, or, it has been said, of the whole of the muscle. It has been stated that these cases, like the traumatic ones, occur with much greater frequency upon the left than upon the right side; but this assertion appears to be very much open to doubt. It is, however, apparently clear that the posterior part of the muscle behind the tendon is most commonly the defective part. It is questionable whether a sac is ever present, though one or two doubtful examples of this certainly rare condition have been recorded. Large masses of the abdominal viscera in the order of frequency named above, namely, stomach, spleen, liver, small intestine, large intestine, mesentery, and rarely duodenum, pancreas, and cæcum, have been found in the pleura. The lung on the affected side is badly developed (it has been said to have been absent), and that on the other side, if the patient has lived, has been found emphysematous.

Fœtuses with this malformation, while quite able to live under the conditions of intrauterine life, seldom survive their birth more than a very short time, because they are unable to carry on aerial respiration. Some, however, have overcome the initial difficulties and have survived for days, months, or even years; but few, if any, have reached adult life. Difficulties in respiration and inability to carry on the processes of nutrition have ended their existence within a comparatively short time of birth. The whole subject therefore belongs more to the domain of the embryologist than to that of the practical physician or surgeon.

Acquired diaphragmatic hernia.—Besides the two forms described, there are others of very infrequent occurrence in which diaphragmatic herniæ have formed through some of the normal openings in the muscle. Sometimes the protrusions have only consisted of fat (fatty herniæ). Sometimes the contents have consisted of some of the abdominal viscera.

Almost all the openings in the diaphragm have been credited with being the occasional seats of herniæ, but it must be owned that many of the accounts sound apocryphal. The most genuine cases have been met with at the two small openings on each side of that portion of the muscle which is attached to the xiphoid appendix, and at the opening for the œsophagus. It would require a good deal of faith to credit the accounts of herniæ occurring at the opening for the vena cava, or those for the splanchnic nerves.

The treatment of these cases, if any, must be on the same lines as that suggested for the traumatic cases.

R. J. G.

CHAPTER L

ACUTE INFLAMMATION OF THE PLEURA

(ACUTE PLEURISY)

PAGE

- Etiology . . . 543
  - Bacteriology of sero-fibrinous pleurisy . . . 545
  - Bacteriology of purulent pleurisy . . . 550
- Morbid anatomy . . . 552
  - Fibrinous or plastic exudation . . . 552
  - Serous and sero-fibrinous exudation . . . 553
  - Purulent effusion (empyema) . . . 554
  - Hæmorrhagic effusion . . . 555
  - Putrid effusion . . . 555
- Effects of pleural effusion . . . 555
- Classification of pleurisies . . . 556
- Acute Pleurisy . . . 556
  - Symptoms . . . 557
  - Physical signs . . . 558
    - *a.* With fibrinous exudation . . . 558
    - *b.* With effusion . . . 559
    - *c.* Stage of absorption . . . 563
  - Course and terminations . . . 563
  - Complications . . . 564
  - Causes of death . . . 566
  - Diagnosis . . . 568
  - Diagnosis of the nature of the effusion . . . 570
  - Prognosis . . . 570
  - Treatment . . . 572
  - Paracentesis . . . 574

(For a brief account of the various forms of Pleurisy from a bacteriological point of view *vide* p. 590.)

PLEURISY

Inflammation of the pleura may occur as a *primary* affection, or i may be *secondary* to some general or local disease.

ETIOLOGY

The influence of *age* and *sex* is not of marked importance, as th disease is met with at all periods of life, but probably occurs wit greater frequency between the ages of twenty and thirty years, an is certainly then more common in males than females.

A *chill* from exposure to cold is very often the immediate exciting cause in this as in other diseases, but as in other diseases it is difficult to state its exact mode of action. Bacteriological research has, however, shown that the susceptibility of an animal to disease may be influenced by lowering its temperature.

The *constitution* and *previous health* of the patient undoubtedly exert a marked effect upon his resisting power to a chill as to other injurious influences, and are factors of importance in determining the occurrence of the disease in any given case.

A *liability to tuberculosis*, particularly of the lungs, undoubtedly predisposes to pleurisy. Inflammation in such cases may be simple, but recent research renders it certain that not only in such patients, but also in individuals presenting no obvious tendency of the kind, an acute pleurisy apparently due to a chill, and occurring in a robust and previously healthy subject, is often of tubercular origin.

The nature of such a pleurisy may even be overlooked on post-mortem examination, as the inflammatory exudation may mask the presence of an acute miliary tuberculosis of the serous membrane. This source of error, however, disappears if the observer is aware of the importance in all cases of examining the condition of the interlobar fissures, the opposed surfaces of which generally become agglutinated at an early period of the disease. There each granulation remains as distinct as when first formed, whereas on the free surface of the pleura they may be effectually hidden by a thick layer of organised exudation.

Acute rheumatism, acute and chronic Bright's disease, cirrhosis of the liver, and malignant disease and cancer, are often complicated with pleurisy. With the exception of the two affections first mentioned, this is not uncommonly due to tubercular infection.

Inflammatory and *other diseases* of neighbouring viscera, particularly of the lungs, pericardium, and mediastinum, are frequently attended by pleurisy, and collections of pus originating elsewhere may also infect or perforate the pleura and set up inflammation.

Pleurisy may also originate from *injury of the chest wall* and the lung, also from a variety of conditions attended by the *presence of septic or other organisms*. Thus septicæmia and the acute fevers, especially scarlet fever and smallpox, may be attended with inflammation of the pleura.

The *bacteriological examination of pleural effusions* has of late thrown much light upon the etiology of the affection, and has led to an attempt to classify pleurisies upon the basis of the micro-organisms which are believed to be the exciting cause of the inflammation.

The following varieties have been described :

1. Pneumococcus pleurisy.
2. Streptococcus pleurisy.
3. Saprogenic pleurisy.
4. Tubercular pleurisy.
5. Staphylococcus pleurisy.

Other organisms, such as the bacillus of typhoid fever, Friedländer's capsuled bacillus, and that of influenza, are also capable of exciting inflammation of the serous membrane, but cases so originating are of less frequent occurrence (*vide* Appendix, p. 590).

We shall in the first place consider the results which have so far been obtained from the bacteriological examination of sero-fibrinous exudations, and subsequently review the evidence as to the nature of the micro-organisms met with in purulent effusions.

Bacteriology of sero-fibrinous pleurisies.—It is generally agreed that in order to form a trustworthy opinion as to the nature of a pleural effusion it is necessary to know—

(*a*) The naked eye appearances.
(*b*) The results of microscopical examination.
(*c*) The effects of inoculation.

It may be stated at the outset that, in the majority of cases of sero-fibrinous pleurisy, no micro-organisms can be found in the effusion, and no results are obtained either from cultures or inoculations.

The subject which has received most attention is the part played by the bacillus of tubercle in the causation of acute pleurisy with an exudation of this character, affecting individuals previously in good health, and often apparently of robust physique and free from hereditary tendency to tuberculosis.

Kelsch and Vaillard[1] examined sixteen fatal cases of pleurisy, in the majority of which the effusion was sero-fibrinous. The subjects were mostly soldiers, in vigorous health before the onset of the illness, which was generally attributed to a chill, and presented all the clinical characters of an acute pleurisy. Death occurred during the course of apparent convalescence. In all the cases there were miliary granulations on the pleura, whilst the lungs were generally free from infiltration, or presented a few recent lesions. The same paper contains an analysis of 113 cases of acute pleurisy, of which nearly 82 per cent. proved to be tubercular. The conclusion of the authors is that 'ordinary pleurisy is only a manifestation of local tuberculosis.' Cases in which the disease is obviously secondary to the specific fevers, or to pneumonia, or appears in the course of acute rheumatism or as a complication of Bright's disease, are excluded from the above very general statement.

Lemoine[2] attempted to obtain cultures in thirty-two cases of acute pleurisy with sero-fibrinous exudation. All the patients were soldiers, and the majority were men previously presenting all the appearances of health, and without hereditary tendency to tubercular disease. Positive results followed in only four cases; in all, the growth was a pure cultivation of *Staphylococcus albus*.

Of the twenty-eight remaining cases, in one the pleurisy was associated with pulmonary tuberculosis, and in fifteen it was followed by that disease. Seven patients left the hospital with suspicious physical signs at the apices of the lungs, and five were apparently cured.

Netter[3] practised inoculation with fluid taken from twenty cases of acute pleurisy occurring in healthy subjects in which the attack was attributed to a chill and was followed by recovery. In eight cases tuberculosis developed in the animal inoculated, whereas in nine cases of acute pleurisy occurring as a complication of acute Bright's disease, cancer, and acute rheumatism, no result was obtained from inoculation.

---

[1] *Archiv. de Physiol.*, 1886, t. ii. p. 162, quoted by Straus, *La Tuberculose et son Bacille*. Paris: 1895, p. 704.

[2] *Bullet. des Hôp.*, 1895, p. 256.

[3] *Ibid.*, 1891, p. 176.

Landouzy,[1] whilst not denying the possibility of acute pleurisy arising from a chill, considers such an event extremely exceptional. He believes that 70 to 75 per cent. of all such cases are of tubercular origin.

Germain Sée [2] is also of opinion that 'so-called simple pleurisy from a chill is only a tuberculous pleurisy, the nature of which has been misunderstood.' He also believes it to be of that nature in the same proportion of cases.

Chauffard and Gombault [3] inoculated twenty guinea-pigs with fluid (3 c.c.) withdrawn by paracentesis from the same number of cases of acute pleurisy, and in ten tuberculosis developed, although tubercle bacilli were not found in the fluid in a single case.

Straus [4] is of opinion that had larger quantities of fluid been used the negative results would have been fewer.

Thue,[5] of Christiania, examined microscopically and attempted to obtain cultures in thirty cases of sero-fibrinous pleurisy. In twenty the results were negative; the ten positive results were as follows:

	Cases
Bacillus tuberculosis	1
Streptococcus	1
Cocci of doubtful nature	2
Staphylococcus aureus and albus	1
S. albus alone	3
S. albus followed by tuberculosis on inoculation	1
Micrococcus cereus	1
	10

In three cases pneumococci were found, but in all the effusion became purulent.

Out of thirty-three cases of sero-fibrinous pleurisy, in which the subsequent state of health of the patient was ascertained, twenty developed tubercle.

Fernet [6] obtained the following results from the bacteriological examination of the effusion in twenty cases of acute pleurisy:—

	Cases
Pneumococcus	4
Staphylococcus	6
Eberth's typhoid bacillus	1
Bacillus tuberculosis	3
No result	6
	20

---

[1] *Rev. de Méd.*, 1886, p. 611.

[2] *Des Malad. Simples du Poumon.*

[3] *Bullet. des Soc. Méd.* Paris: 1886, pp. 441 and 518; *des Hôp. de Paris*, 1884, p. 309.

[4] *Op. cit.*, 1895, p. 705.

[5] Vide *Bullet. des Hôp.*, 1895, p. 439.

[6] *Ibid.*, 1895, p. 145.

The cases recorded by this observer are not all, however, such as would be termed idiopathic, or due to chill, as three of the cases of pneumococcal pleurisy were probably secondary to pneumonia. He also obtained positive results in a larger proportion of cases than most other observers.

Dr. Washbourn[1] has drawn attention to the fact that in acute pleurisy caused by the pneumococcus the constitutional symptoms may resemble those of pneumonia—viz. a sudden onset with rigor, labial herpes, high fever, cough, rapid breathing, and delirium. In the three cases described the effusion was purulent and contained the pneumococcus.

Similar cases were mentioned by Dr. Hale White in the discussion which followed the reading of Dr. Washbourn's paper.

We are indebted to Dr. Sidney Martin for the following account of seven cases of acute pleurisy with fever, in which the effusion was tested by bacteriological methods :

—	Nature of fluid	Bacteria found	Toxicity of fluid in rabbits
Case 1.	Yellowish : clots after removal	Not examined for bacteria	Intra-venous injection was followed by a slight rise of temperature
Case 2.	Slightly turbid : does not clot	Pure growth of streptococcus	Intra-peritoneal injection followed by high fever and death. Pure growth of streptococcus obtained from dead animals
Case 3.	Clear : clots after removal	No bacteria (agar, broth, gelatine)	No evident toxicity of fluid
Case 4.	Clear : clots feebly	Pure growth of cocci, chiefly diplococci	No evident toxicity from one intra-peritoneal injection
Case 5.	Clear : clots slowly but firmly	No growth (agar)	—
Case 6.	Clear : does not clot	No growth (agar)	—
Case 7.	Clear : clots imperfectly	Pure growth of staphylococci (same cocci in sputum of patient)	Intra-peritoneal injection of 15 c.c. caused fever

All the cases ended in recovery except Case 2, which was rapidly fatal ; the fluid became purulent, and peritonitis supervened.

---

[1] *Med.-Chir. Trans.*, 1894.

Osler[1] examined the post-mortem records of the Johns Hopkins Hospital in order to determine the proportion of cases of pleurisy of tubercular origin, with the result that in 101 successive cases, from his own wards, only thirty-two were found to be of that nature.

This is in marked contrast with the statements of the French observers already quoted, from which the percentage would appear to be from 70 to 75.

Evidence of this kind as to the general incidence of tuberculous pleurisy is, as Osler points out, of greater value than mere clinical impressions. But in this, as in other cases, it must be accepted with the proviso that the post-mortem reports must contain a positive statement as to the absence of tuberculosis. An analysis of such records, with a view to determine the incidence of a lesion which was not specially looked for at the time, is, in the experience of the writer, rarely trustworthy. Hardly a single note of some condition may appear up to a certain date, whereas subsequent investigation may prove it to be of common occurrence. It has so often happened to us to demonstrate the unsuspected tubercular nature of a pleurisy by an examination of the interlobar septa, that we have come to mistrust all records from which mention of them is omitted.

Grancher[2] states that in cases of acute pleurisy with sero-fibrinous effusion a tubercular origin may be suspected from the presence of certain physical signs pointing to previous disease of the apex of the lung of the affected side. A skodaic note on percussion, with increase of vocal fremitus, is found as in non-tubercular cases with considerable effusion; but in addition the breath-sounds are feeble, but at the same time harsh in quality, or simply harsh.

The inference that a large number of cases of acute pleurisy, apparently idiopathic or due to a chill, are really of tubercular origin, is still further strengthened by an examination of the subsequent history of the patients.

Fiedler[3] found in ninety-two cases of acute pleurisy, in which the effusion was proved by aspiration to be sero-fibrinous, that at the end of two years twenty-eight were dead from pulmonary tuberculosis, whilst of the remainder only twenty-one appeared to be healthy. The others either had tubercular disease of the lungs, or presented physical signs suggestive of its presence.

Barrs[4] investigated the subsequent history of sixty-two cases of acute pleurisy observed during the years 1880–84, at the Leeds Infirmary. Six years later twenty-two of these patients were found to have died of tuberculosis.

Richochon[5] ascertained the subsequent history of thirty-three cases of acute pleurisy, and found that fourteen had died of tuberculosis; whilst in seven, either before or after the attack, there had been some manifestation of that disease.

---

[1] Shattuck Lecture on *Tuberculous Pleurisy*, 1893.

[2] *Malad. de l'App. Respir.* Paris: 1890, p. 292.

[3] Volkmann's *Sammlung klin. Vorträge*, 1882.

[4] *Brit. Med. Journ.*, 1890, vol. i. p. 1059.

[5] *Etud. Expér.* Verneuil, 1887, t. ii. p. 573.

Bowditch (jun.),[1] from a similar inquiry amongst the patients suffering from pleurisy seen by his father in private practice during thirty years (1849–79), obtained the following results:

—	Cases	Dead from pulmonary tuberculosis	Dead from some other disease	Doubtful	Alive and healthy	Percentage of tuberculous cases
1849–59 .	30	12	5	2	11	43·3
1860–69 .	19	9	3	—	7	47
1870–79 .	41	9	6	1 suffering from tuberculosis	25	24

The smaller number of tuberculous cases in the last decennial period may be due to the shorter time which had elapsed since the attack of pleurisy.

The following *conclusions* appear to be justified by the evidence adduced:

1. That in a very large proportion of cases of acute pleurisy with sero-fibrinous exudation, no results are obtained from the bacteriological examination of the exudation.

2. Tubercle bacilli may not be found in cases which are undoubtedly of that nature, and, as a rule, they are only present in the coagulated portion of a sero-fibrinous effusion.

3. The inoculation of fluid from a case of tubercular pleurisy into susceptible animals may not be followed by tuberculosis.

4. That, nevertheless, the bacillus of tubercle is the causative agent in a very large proportion of cases of sero-fibrinous pleurisy, and that this is true when the attack is apparently simple, idiopathic, and attributed to a chill.

5. That when no bacteriological examination has been made, or when no organisms have been discovered, prolonged observation proves that in many cases pulmonary tuberculosis subsequently develops.

6. That by the use of bacteriological methods of diagnosis it is possible in some cases to determine the nature of a sero-fibrinous effusion.

The percentage of tubercular cases (70–75) given by the French observers above quoted may be too high, but the writer has long been convinced that tubercle plays a more important part in the causation of pleurisy than is generally believed; and Osler states,[2] 'I confess that the more carefully I have studied the question the larger does the proportion appear to be of primary pleurisies of tuberculous origin.'

---

[1] *Med. News*, 1889, vol. lv. p. 63.

[2] *The Principles and Practice of Medicine*, 2nd edition, 1895, p. 593

The appearance of pulmonary tuberculosis after an attack of pleurisy does not, of course, prove that the pleurisy was tubercular; but its occurrence in such a large number of cases is presumptive evidence which cannot be ignored.

By some observers, however, the fact is denied.

Osler states that 'the subsequent history of cases of acute pleurisy forces us to conclude that in at least two-thirds of the cases of tubercular pleurisy it is a curable affection.'[1]

It must also be borne in mind that the course of tuberculosis of the serous membranes is far more favourable than when the disease attacks the lungs and other organs. Of this the clinical history of tubercular peritonitis affords a striking example. The inference that in many cases of tubercular pleurisy the disease is arrested at the time and never reappears is therefore justified, and is supported by pathological experience.

Bacteriology of purulent pleurisies.—The nature of the micro-organism which is presumably the exciting cause of the inflammation can be determined in a much larger proportion of cases of pleurisy with purulent than with sero-fibrinous effusion.

Those most commonly present are the pneumococcus, staphylococcus, the streptococcus albus, saprogenic organisms, and the bacillus of tubercle. Friedländer's capsuled bacillus, the typhoid bacillus, and that of influenza, and certain others are also probably capable of producing a purulent effusion.

Usually one organism only is present, but two or more may be associated, *e.g.* pneumococci with streptococci or staphylococci, or the tubercle bacillus with staphylococci, streptococci, or with various organisms of putrefaction.

An effusion may be purulent from the beginning, or it may be primarily sero-fibrinous, or serous, and may acquire the purulent character at a later period. Streptococci may be present before the effusion has become purulent.

Netter[2] found the staphylococcus pyogenes alone in but a single case of purulent pleurisy, and this was secondary to ulcerative endocarditis. He is of opinion that when it is discovered in such effusions it is not the cause of the inflammation, which is due to the associated organisms, *e.g.* streptococci, pneumococci, or saprogenic bacteria; also that if only staphylococci are readily found the case is probably tubercular, more time being required to determine the presence of tubercle bacilli than of the other organisms mentioned.

A. Fraenkel states that when no micro-organisms are found in a purulent effusion the presumption is strongly in favour of the case being tubercular, and suggests that the virus may be present in the form of spores.

Positive results may be obtained from inoculation when tubercle bacilli are not found on microscopical examination. Thus Netter produced tuberculosis in guinea-pigs in twelve out of thirteen tubercular empyemata; and Straus,[1] to whose work we are indebted for many of the references here given, obtained similar results in six cases. He points out that by boiling and renewing the staining fluid (Ziehl's) several times, the presence of tubercle bacilli in purulent effusions may be more certainly determined.

---

[1] *The Principles and Practice of Medicine*, 2nd edition, 1895, p. 593.

[2] *Bullet. des Hôp.*, 1890, p. 441.

Ehrlich states that since 1888 he has never failed to find bacilli in tubercular empyemata, but they are often present in very small numbers, and prolonged search may be necessary.

Thue,[2] as the result of a bacteriological examination of the effusion in twenty-four cases of purulent pleurisy, found—

	Cases
Pneumococcus	14
Streptococcus	5
Tubercle bacillus	3

The pneumococcus cases were chiefly observed during epidemics of influenza. Twelve out of the fourteen recovered; in one case death was due to mitral regurgitation with cardiac failure; in the other to pneumonia.

Netter,[3] from the examination of 109 cases (apparently 110, *vide infra*) of purulent pleurisy, obtained the following results:

	Cases
Streptococcus	51
Pneumococcus	32
Saprogenic bacteria	15
Tubercle bacillus	12
	110

The incidence in children and adults was as follows:

Nature of micro-organism	Percentage	
	Children	Adults
Streptococcus	17·8	53
Streptococcus and pneumococcus	3·6	2·5
Pneumococcus	53·6	17·3
Staphylococcus	—	1·2
Tubercle bacillus and others	14·3	—
Organisms of putrefaction	10·7	8
Tubercular and organisms of putrefaction	—	25

It will be seen from the above results, which are in accord with those of other observers, that the percentage of tubercular cases is much smaller in purulent than in sero-fibrinous pleurisies; also that in children more than half the cases of empyema are due to pneumococcus infection, whilst in adults in exactly the same proportion they are of streptococcus origin. A brief account of the various forms of pleurisy from a bacteriological point of view will be found at the end of Chapter LII. (*vide* p. 590).

---

[1] *Op. cit.*, p. 716.

[2] *Bullet. des Hôp.*, 1885, p. 439

[3] *Ibid.*, 1889, p. 441.

MORBID ANATOMY

1. **Fibrinous or plastic exudation.**—The earliest change observed in pleurisy induced experimentally, is an injection of the vessels of the subserous layer. The membrane then loses its polish; the nuclei of the endothelial cells of the pleura undergo proliferative changes, and the stomata of the membrane and the neighbouring lymphatics become blocked with fibrin and corpuscles, and form fine projecting granulations.

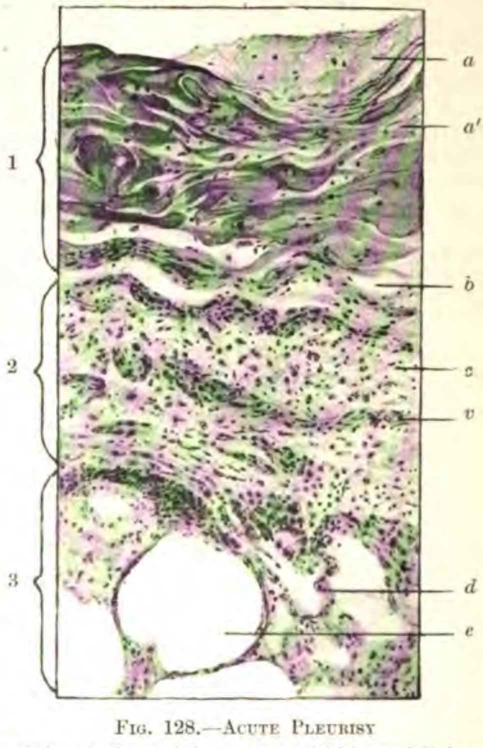


FIG. 128.—ACUTE PLEURISY

Showing—1, exudation; 2, pleura; 3, lung; *a*, superficial layer of fibrinous exudation; *a'*, deeper and more laminated layer of exudation; *b*, spaces occupied by fluid; *c*, cellular infiltration of pleura; *v*, injected vessel; *d*, wall of superficial alveolus slightly thickened; *e*, emphysematous air vesicle.

A fibrinous exudation, enclosing in its meshes leucocytes and red blood corpuscles, appears upon the surface, and as the cellular and fibrinous elements of the exudation increase in quantity a layer of coagulated lymph of varying thickness is formed. The fibrin lying nearest to the pleura is arranged in dense laminæ, more superficially it forms trabeculæ in the meshes of which is a finer

DESCRIPTION OF PLATE III.

CELLS FROM CASES OF CLEAR INFLAMMATORY PLEURITIC EFFUSION, ILLUSTRATING THE SHAPES AND SIZES WHICH ARE COMMONLY MET WITH.

Drawn with an oil immersion lens (one-twelfth). The size of the cells may be estimated by comparing them with the red corpuscles seen in fig. 1, *b c*. Fig. 1 is unstained; fig. 2 was stained with hæmatoxylin and carmine; fig. 3 with hæmatoxylin and eosin.

Plate III.

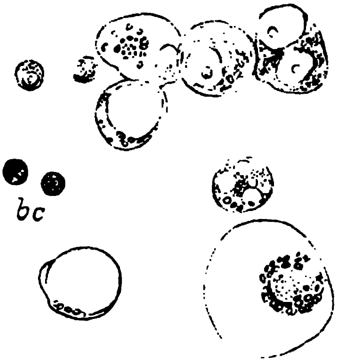


Fig. I.

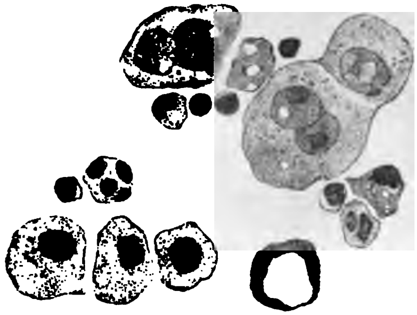


Fig II.

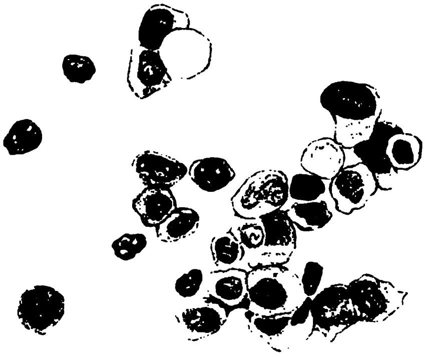


Fig. III.

Bale & Danielsson, L

network of fibrin enclosing corpuscles. If this is stripped off, an injected surface is exposed, and in septic cases this may be dotted with punctiform extravasations. Both layers of the pleura are usually affected at the same time, but the change may be more marked on the visceral aspect, owing doubtless to the fact that in the majority of cases infection occurs from the lung. The inflammatory process may cease at this stage; if so, the case is spoken of as one of dry pleurisy. The exudation, as a rule, then becomes vascularised and transformed into connective tissue, and, as a rule, the two surfaces are united.

The limited adhesions so commonly found post-mortem probably originate in pleurisies of this type. The exudation may, however, be absorbed without the formation of adhesions, some opacity of the visceral pleura alone remaining to mark its site.

Microscopical examination of a section of an inflamed pleura often shows that, in addition to the changes above mentioned, some degree of thickening of the interlobular septa has occurred.

When of tubercular origin the layers of exudation upon the surface of the membrane may become very thick and stratified, and from prolonged rubbing they may acquire a rough, coarsely granular appearance.

Serous and sero-fibrinous exudation.—When effusion occurs, serous fluid is poured out between the layers of fibrin which adhere to the surfaces of the membrane, and is thus, for a time at least, contained in a species of sac. Flocculi, flakes and larger, often rounded, fibrinous coagula may be found floating free in the fluid or they may have sunk to the lower part of the cavity, whilst in some cases fine meshes of fibrin traverse it and enclose the effusion in incomplete loculi, the whole presenting a somewhat gelatinous appearance.

The fluid is of a yellowish or faintly green tint, alkaline in reaction, and of a specific gravity which may vary between 1·005 and 1·030 (Fraentzel), but is most commonly between 1·016 and 1·023 (Mehu). If the corpuscular elements are few, it is clear and translucent; if they are more abundant, it is opalescent, cloudy or slightly turbid. Leucocytes, either unchanged or in various stages of transformation into pus cells, larger cells derived from the proliferation of the endothelium, red corpuscles in varying numbers and fibrin, may be recognised on microscopical examination (*vide* Plate III.). The fluid is, as a rule, highly albuminous, but the amount of albumen varies considerably (31 to 77 per cent., Walshe). After withdrawal of the fluid by paracentesis the contained fibrin may coagulate spontaneously, or only after standing for a long time exposed to the air. Urea, uric acid, sugar, cholesterin, leucin, tyrosin and xanthine have all been found in effusions of this character. The quantity of fluid effused varies from a few ounces to many pints. From three to four pints of serous fluid are often withdrawn from the pleura at a single aspiration, and as much as 280 ounces of fluid have been so removed.

Purulent effusion (empyema).—The factors which probably determine the character of an inflammatory effusion in the pleura are the virulence of the micro-organisms present and the degree of resisting power of the patient. Marked activity of the morbid process is accompanied by an exudation of fluid rich in cell elements and deficient in fibrin; under such circumstances it may assume the purulent character almost from the outset. This is especially frequent in streptococcus pleurisies. An effusion of this nature may, however, be primarily sero-fibrinous or sero-purulent; it is then often merely turbid and contains fibrinous flakes. It may be found post-mortem to have separated into layers, of which the upper is clear and of a greenish tint, whilst the lower part of the cavity contains thick yellow creamy pus and masses of coagulated lymph. In the most acute cases it consists of thin, watery, yellow pus and little or no fibrinous coagulum is adherent to the surface of the serous membrane, its normal shiny appearance being almost completely retained. The fluid is usually neutral in reaction and free from odour, but a purulent effusion may be acid, and sometimes, either from admixture with air or from the association of gangrene of the lung or independently of any obvious cause, it may become putrescent and acquire a fœtid odour.

In empyemata of long standing the pleura is often much thickened and the effusion inspissated, the formed elements being in process of fatty degeneration. At a still later period, when much retraction has taken place, the walls of the sac are thickened and may be of fibroid structure and almost cartilaginous consistence. The fluid has then, as a rule, been either partially or completely absorbed and the contents are either pale yellow, firm and curdy, or some milky-looking fluid may still be present. Crystals of cholesterin and of the fatty acids have been found under such circumstances.

Calcified masses, generally in the form of plates, situated most frequently at the base of the cavity and often upon the diaphragm, are produced by deposition of lime salts in an exudation which has passed through these stages. In the great majority of such cases the primary disease has been tubercular.

An empyema may be **loculated** from the presence of septa, which, in the form of adhesions, may have existed prior to the acute attack, or they may have been formed during it.

The appearance of 'loculation' is not confined to empyema, as in sero-fibrinous effusions the fluid may be separated into sacs by masses of coagulated lymph. Loculated effusions and such as are encapsuled or encysted are most common at the base, but they may be found almost anywhere. When situated between the interlobar septa, or between the diaphragm and the lung or between the latter and the mediastinum, the diagnosis of the condition or the discovery of the exact site of the effusion is often a matter of great difficulty. The character of the fluid in separate sacs may differ; in one it may have become purulent, whilst in another it still remains serous.

Hæmorrhagic effusion.—This variety of effusion is, on the whole, rare. It is met with in cases of cancer and tuberculosis, but may occur independently of either of those diseases.

It is not uncommon in association with Bright's disease and cirrhosis of the liver. In a case of the latter disease recently under the care of the writer, seven pints of fluid, so closely resembling blood as to cause a moment's hesitation as to its real nature, were withdrawn from the right pleura.

In another, a case of double pleurisy following pneumonia, and almost certainly due to pneumococcus infection, the fluid in the left chest was purulent, whilst in the right it closely resembled blood. The left chest was drained and the right aspirated; the patient made a good recovery.

In very malignant types of the specific fevers, pleurisy may be attended with a hæmorrhagic effusion.

Putrid effusion.—An effusion may be of this nature from the entrance of putrefactive organisms from the lung, as in cases of gangrene, or the gangrenous change may be primary in the pleura.

In pneumothorax a fœtid effusion is occasionally found, but fœtor may be present apart from either gangrene or the entrance of air into the cavity. A septic pulmonary infarction may also give rise to a putrid pleurisy, and cancer of the œsophagus with rupture into the pleura may be similarly complicated. Pleurisy accompanying septicæmia, or due to perforation by pus derived from an abscess in connection with the stomach or intestines, may also be attended with an effusion of this nature.

A putrid effusion generally consists of thin, dirty-yellow turbid pus, of a sour offensive odour, which is perceived immediately the cavity is opened.

Effects of a pleural effusion.—The effect upon the lung and other organs varies to some extent with the amount of the effusion and the presence or absence of positive intra-thoracic pressure.

But even with a slight effusion occupying the base of the sac, the part of the lower lobe in contact with the fluid is generally completely collapsed. With large effusions the whole lung may be found airless, bloodless, and of a slate-grey tint, lying close to the spine and surrounding its root attachments.

The heart, with the mediastinum, is displaced towards the sound side, unless retained in position by adhesions. This occurs at an early period, and increases *pari passu* with the effusion, whereas displacement downwards of the diaphragm or liver is always a sign of positive intra-pleural pressure (Douglas Powell). The heart does not undergo axial rotation, but the apex may be slightly lifted or the heart may lie more transversely than normally.

Neither the displacement of the heart nor the collapse of the lung is due in the first place to pressure. The former results from the loss of the normal elastic traction of the affected side, and the latter from the absence of inspiratory enlargement of the thorax, combined with the natural elasticity of the lung, which causes it to retract as the fluid accumulates in the closed sac of the pleura.

The collapse of the affected part of the lung is rendered complete by the absorption into the blood of the air which it contained (*vide* Collapse, p. 288).

CLASSIFICATION OF PLEURISIES

In considering the etiology of the disease reference has been made to the attempt to classify pleurisies on the basis of the micro-organisms present in the effusion; owing, however, to the negative results obtained in so many cases, it is impossible at present to utilise this classification for clinical purposes.

The varieties of the disease are so numerous, and an elaborate classification is of such little practical utility, that we shall be content to enumerate the descriptive terms in common use. The course of the disease affords a basis for dividing cases into *Acute* and *Chronic*. Pleurisy arising independently of any other disease is described as *Primary* or *Idiopathic*; if it occurs as a complication of some other affection, as *Secondary*.

According to the nature of the exudation a pleurisy is spoken of as *dry*, *fibrinous*, *plastic* or *adhesive*, or as pleurisy *with effusion*.

The varieties of the effusion give rise to the terms serous, sero-fibrinous, sero-purulent, purulent (empyema), hæmorrhagic and putrid.

The terms *traumatic*, *diaphragmatic* (or phrenic), *cancerous* and *tubercular* do not need explanation.

In this work the subject will be further considered under the following headings:

ACUTE PLEURISY
CHRONIC PLEURISY
TUBERCULAR PLEURISY
DIAPHRAGMATIC PLEURISY

ACUTE PLEURISY

Under this heading we shall describe all forms of acute inflammation of the pleura irrespective of the nature of the accompanying exudation.

It is more in accordance with custom, and is possibly in some respects more convenient, to deal with the subject of Purulent Effusion or Empyema separately, but by so doing the fact that every case of empyema has been at one time a case of acute pleurisy is apt to be lost sight of, and that method of treating the subject tends, we believe, to foster the view, which certainly exists in the minds of students, that empyema is in some unexplained manner an affection entirely distinct from acute pleurisy.

SYMPTOMS

The *onset* is usually acute, and accompanied by severe pain in the side. There may be a single rigor, as in pneumonia, and in pneumococcus pleurisy the attack, as regards the mode of invasion and the general symptoms, may for a time closely simulate that disease. More often, however, the rigor is less severe, but shivering is repeated. Rigors are absent in a considerable number of cases, and in children they may be replaced by vomiting, headache, or convulsions. The pain is increased by breathing or cough, and by any act which causes movement of the affected side; it is generally stabbing or dragging in character, and referred to the infra-mammary or lower axillary region. More rarely it is localised in the epigastric or scapular region. It is usually most severe at the outset and diminishes as effusion occurs. Pain may occur independently of signs of friction of the inflamed surfaces, or it may be entirely absent. Its severity is chiefly influenced by the state of the nervous system and the intensity of the inflammation. Tenderness on pressure is also experienced in some cases.

Cough is usually present, and as a rule is worse during the early stages of the attack. It is short and dry, and is suppressed as much as possible because it increases the pain. If bronchial catarrh coexists, there may be mucoid expectoration, and with pneumonia the sputa may be rusty.

A copious expectoration of watery fluid points to œdema of the opposite lung; it may, however, appear without signs of such a condition and even in dry pleurisy.[1]

In some cases a very copious albuminous expectoration accompanies pleurisy; this is especially observed after the fluid has been withdrawn by paracentesis.

The *decubitus* varies; during the early stages the patient usually lies on his back or on the sound side. Walshe states that lying on the affected side generally increases the pain, but it is not uncommon to find a patient lying on his back with the body bent towards the affected side. After considerable effusion has occurred the patient probably more often lies upon the affected side than in any other position. With double effusion the dorsal position is assumed, whilst in diaphragmatic pleurisy the patient either sits up or leans forward.

Dyspnœa.—The frequency of breathing is almost invariably increased, although the patient may not be conscious of dyspnœa; usually, however, the two are associated. In children the number of respirations may amount to fifty or even many more per minute.

Very rapid, shallow breathing in the early stages is due to the restriction of the thoracic movements from a fear of increasing the pain. At a later period the degree of dyspnœa and the rate of respiration are usually in proportion to the amount of the effusion and to the rapidity with which the fluid is poured out, but the dyspnœa is generally less severe after effusion has occurred. The presence of cardiac, bronchial, or pulmonary complications, but particularly of pericarditis or a double pleural effusion, tends greatly to increase the dyspnœa.

---

[1] Ferrand, quoted by Wilson Fox. *Op. cit.*, p. 977.

The *pulse* is quickened, and may be 100 to 120, but there is not the marked alteration in the pulse-respiration ratio characteristic of pneumonia. Walshe states that in an uncomplicated case in a male he has never observed a ratio lower than 2 to 7.

The *temperature* for the first week or ten days generally ranges between 101° and 102°, but with the pneumonic type of onset it may reach 104°, and may continue for a week at a high level, an incomplete crisis occurring about the seventh day followed by a subsequent rise. In some cases fever may be present before the local signs become obvious. The fever as a rule declines by 'lysis,' rarely by 'crisis,' with its attendant phenomena; the evening rise of temperature gradually diminishes, and in the morning the temperature is normal.

A sero-fibrinous exudation persisting for many weeks may be accompanied by a typical hectic temperature suggestive of the formation of pus. Such cases are probably tubercular, but recovery of health may ultimately be complete, and the fluid may be absorbed. Some dulness, however, usually remains at the base, due probably to the incomplete removal of the solid portions of the effusion and to thickening of the pleura.

The *general symptoms* are those which usually accompany an acute illness. The skin is hot and dry, but usually becomes moist at a later period when effusion has occurred. There is rarely delirium, except in cases of pneumococcus pleurisy simulating pneumonia. If, however, the effusion is overlooked the patient may after a time pass into a condition somewhat resembling the typhoid state with delirium of a low muttering type. We have known this to occur when the post-mortem examination showed that the effusion was still sero-fibrinous. The *urine* presents the usual febrile character; it is diminished in quantity, dark in colour, of a high specific gravity, and deposits lithates.

PHYSICAL SIGNS

(*a*) **Of pleurisy with recent fibrinous exudation.**—On inspection the movements of the affected side are seen to be restricted, and the patient may be lying with the body bent towards that side. On palpation the vocal fremitus is usually normal. There may be diminished resonance on percussion from incomplete expansion of the lung. On auscultation the breath sounds are weak. The typical and diagnostic sign is a friction sound, possibly audible on deep inspiration only. Often, however, in children and sometimes in adults it is not heard. It usually consists of a succession of sounds of a grating character, but may resemble a superficial crackling râle. Friction fremitus is rarely if ever felt at the onset of an acute attack, owing to the restricted movements of the affected side.

A to-and-fro friction sound of cardiac rhythm and simulating a pericardial rub may be present, but is more commonly observed in the less acute forms of pleurisy which occur during the course of pulmonary tuberculosis. The vocal resonance is usually unaltered.

(*b*) **Of pleurisy with effusion.**

Inspection.—If there is no pain, as is not uncommonly the case now that the layers of the pleura are no longer in contact, the movement of the affected side may for a time be less restricted than before the occurrence of effusion; but, as the fluid increases in quantity, expansion diminishes and with large effusions is altogether wanting. When the fluid is limited in amount and basic in site the upper part of the affected side may still expand to some extent.

The chest may appear smooth and rounded from obliteration of the intercostal depressions, but with sero-fibrinous exudation actual bulging of the spaces is very rare. They may recede during inspiration, even when there is a considerable effusion.

The cardiac impulse may be seen to be displaced towards the sound side. For an equal amount of effusion this is more marked in left-sided than in right-sided pleurisy.

The expansion of the unaffected side is generally increased.

Pulsation of a pleural effusion is a sign rarely observed, and is almost limited to left-sided empyemata. It has, however, been noticed in a case of sero-fibrinous effusion, and occasionally with a right-sided empyema; but of forty-two cases collected by Osler only three were on the right side. Pulsation may also occur in cases of pyo-pneumothorax. It is to be distinguished from the pulsation, synchronous with the heart, of an abscess sac resulting from the perforation of the chest wall by an empyema. The impulse may be heaving and is generally situated about the sternal region, but may be diffused and most marked near the angle of the scapula.

On *palpation* the position of the cardiac impulse, a most important point, may be more accurately determined. The vocal fremitus is as a rule either absent or greatly diminished over the site of the fluid; it may, however, although rarely, remain unaffected. This may be due to previous adhesions between the pleural surfaces, or possibly in some cases to conduction of vibrations from the spine along the ribs and integuments.

Fluctuation in the intercostal spaces can rarely be elicited, even in purulent effusions.

Œdema of the affected side is seldom met with except in empyema of long standing, but it has been observed in serous effusions. It appears to be of more frequent occurrence in children than in adults, and in very acute septic cases at any age.

On *percussion* there is dulness over the site of the effusion, the note elicited being absolutely flat and wanting in tone, whilst the sense of resistance is much increased. In cases of moderate effusion the dulness is almost always most marked at the base. Its upper level is not horizontal; as a rule it rises highest in the axilla and gradually slopes backwards and forwards, leaving in front beneath the clavicle a triangular area, where the note is of amphoric or of tympanitic quality (skodaic resonance), and behind an area similar in shape close to the spine, in which, although the note is dull, it is not so completely toneless as at the base. This line is sometimes spoken of as Damoiseau's curve.

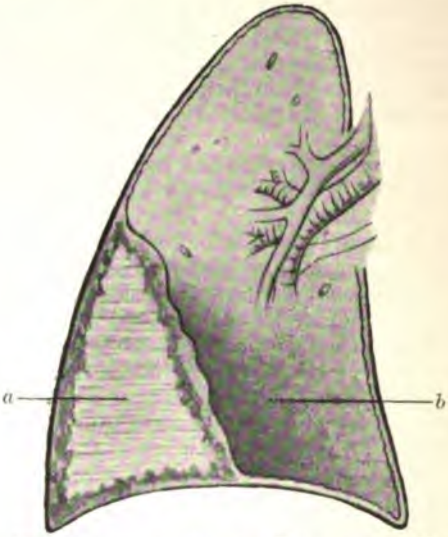


FIG. 129.—DIAGRAM ILLUSTRATING THE EXUDATION OF FLUID WITHIN A SAC LINED BY COAGULATED FIBRIN

a, sac containing fluid; *b*, collapsed lung.

The level of dulness is but little, if at all, influenced by the position of the patient. This is owing to the fact that the surfaces of the pleura, except where separated by the fluid, are in close contact, and thus a sac is formed which exactly contains the fluid. It is obvious that if the fluid were free to move as the patient altered his position, say from sitting up to lying down, a portion of lung previously collapsed must expand and another must undergo partial collapse.

The dulness may extend beyond the middle line of the sternum if the effusion is considerable, and will then merge into that of the displaced heart.

Auscultation.—The respiratory sounds are weak or absent below the level of the fluid; but in exceptional cases in adults, and more often in children, tubular breathing is audible over the whole of the dull area. *This is a frequent source of error in diagnosis.*

Cavernous or even intense amphoric breathing, sometimes associated with gurgling râles, may also be heard, and may lead to the diagnosis of excavation of the lung or of pneumothorax. These signs have been observed at the apex and elsewhere in association with skodaic resonance on percussion above the margin of the fluid.

Voice sounds.—Vocal resonance is usually absent below the level of the fluid. A peculiar modification of the voice is often present, generally with effusions of a moderate amount, to which the term *ægophony* is applied. It is a high-pitched sound of a tremulous, twanging character, likened to the bleating of a goat or the human nasal voice. It is generally stated to be most commonly present about the angle of the scapula, or at the upper level of the liquid where there is only a thin layer, sufficient to modify but not to suppress the voice. Dr. Frederick Taylor,[1] from an analysis of twenty-one carefully observed cases, found it in nine of them to occupy a small area below the angle of the scapula; in four it was heard over a band-shaped area extending from the spine towards the axilla, and always far below the upper margin of the dulness, and in eight it was spread over a large area at the base. In six of these the dulness extended some inches above the upper limit of ægophony.

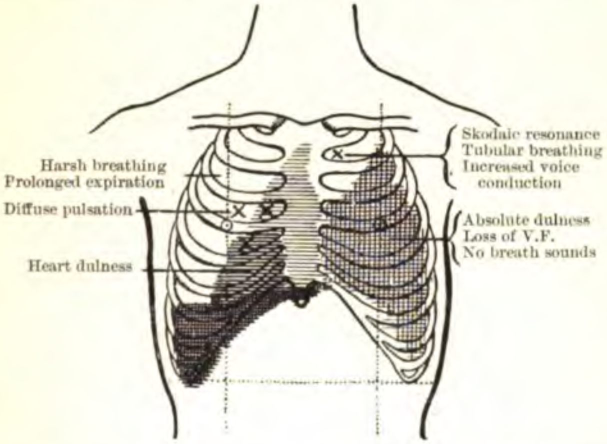


FIG. 130.—SHOWING THE PHYSICAL SIGNS IN A CASE OF LONG-STANDING LARGE EFFUSION INTO THE LEFT PLEURA

The exact mechanism by which this sound is produced is not clearly understood. Dr. Stone stated that the sound was due to 'the suppression of the fundamental note and of the lower harmonics of the vowel sounds, while the higher harmonics are transmitted through the fluid in an accentuated form.' Dr. Taylor concludes that it is merely a discordant modification of the voice due to altered conditions of vibration in the bronchial tubes, and that the presence of fluid is not necessary for its production.

---

[1] *Med. Chir. Trans.*, vol. 28.

The vocal resonance is, as already stated, usually lost except at the upper margin of the dull area, but the whispered voice may be clearly transmitted. Bacelli considers that by the presence or absence of this sign it is possible to differentiate a serous from a purulent effusion, the latter fluid, owing to its being thicker and containing more formed elements, preventing the conduction of the sound. 'Aphonic pectoriloquy' may, however, be met with in sero-fibrinous and purulent effusions.

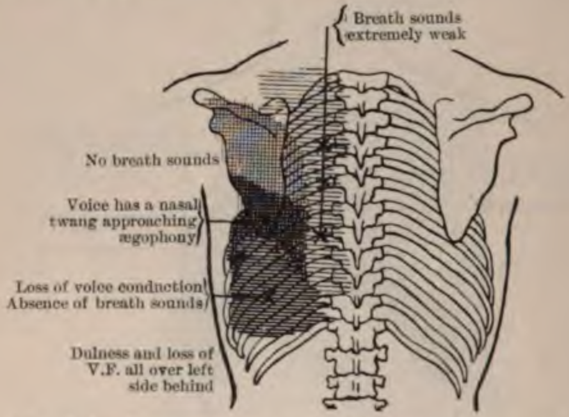

Breath sounds extremely weak
No breath sounds
Voice has a nasal twang approaching ægophony
Loss of voice conduction Absence of breath sounds
Dulness and loss of V.F. all over left side behind

FIG. 131.—SIGNS PRESENT ON THE POSTERIOR ASPECT OF THE CHEST IN THE SAME CASE

Mensuration.—Measurement, by means of the cyrtometer, shows that in the majority of cases the area of the chest is enlarged, the unaffected side being also increased, but to a lesser degree. The enlargement begins below by a widening of the intercostal spaces and a more horizontal position of the ribs—changes which give to the chest a rounded form. It is stated that the sternum is moved towards the affected side, the deviation being most marked in the lower part of the thorax (Peyrot quoted by Wilson Fox).

Positive intra-thoracic pressure is indicated by (*a*) decided increase in the measurement of the affected side, and the extension of the dulness above the third cartilage; (*b*) the Skodaic resonance beneath the clavicle becoming either tubular or disappearing; (*c*) downward displacement of the diaphragm, as shown by the absence of the stomach note over the sixth rib in the left nipple line, or by depression of the liver; (*d*) a displacement murmur may be present (Douglas-Powell).

(*c*) **Stage of absorption.**—The changes in the physical signs which attend the absorption of the fluid are: diminution of the enlargement, as determined by the cyrtometer; movement of the displaced heart towards its normal position; the gradual disappearance of the dulness from above downwards; and the reappearance of the respiratory sounds over the previously dull area. Ægophony may return, and a redux friction sound generally becomes audible. Friction fremitus is also not infrequently present at this stage. Some dulness and diminished vocal fremitus, with weak breathing, often remains for a time at the base, owing to the longer period which is required for the absorption of the more solid portions of the exudation; but in favourable cases, after a time, these signs disappear, and the re-expansion of the lung is complete.

COURSE AND TERMINATIONS

The course of the affection is very variable. If there is no effusion the inflammation may speedily subside, and the patient may be well in a week. An effusion in many cases continues to increase in quantity for about a fortnight or sixteen days; the inflammatory process then ceases, and that of absorption commences and proceeds without interruption. In others there is no increase after the end of the first week; then there follows a short period during which no change is noticed in the physical signs; after this the percussion note indicates that absorption is in progress. The absorption of an effusion during the continuance of considerable pyrexia is a rare event, but we have observed its occurrence in a case which was probably of tubercular origin. If fluid is poured out, even in moderate quantity, recovery is rarely complete in less than a month or more; whilst if the amount is considerable, and particularly if it becomes necessary to perform paracentesis, the illness will be of much longer duration. Cases are, however, recorded in which large effusions have been rapidly absorbed, and others in which fluid has remained for more than a year and has even then proved to be still serous. Occasionally a rapid disappearance of fever follows the removal of the fluid by paracentesis.

If the fluid is purulent and the operation for empyema is necessitated, the duration of the illness may be greatly prolonged. The nature of the effusion and the degree of thickening of the visceral layer of the pleura, due either to the organisation of adherent coagulated lymph, or to changes in the membrane and the interlobular septa, are the chief factors which determine the extent of re-expansion of the lung, and consequently of the retraction remaining after the fluid has been absorbed. The results following paracentesis prove, however, that re-expansion may be complete after an effusion has been present for very long periods, even for six months or more.

Purulent effusions, if of pneumococcal or tubercular origin, may be absorbed; but such an event is hardly to be expected in streptococcus pleurisies, and is, under all circumstances, of rare occurrence. The caseous collections surrounded by thick walls, with possibly some watery-looking fluid still remaining, which are occasionally found post-mortem at the base or between the lobes, are probably examples of this condition. Some degree of retraction often remains for a time, even in the less severe cases, but in these complete re-expansion may ultimately occur. Retraction is much more marked and persistent when absorption has been long delayed, owing generally to the fact of the effusion having been purulent. Such a condition is not, however, incompatible with health, as compensatory enlargement of the opposite lung, accompanied by increased functional activity or more complete expansion of the upper lobe of the affected lung, may restore equilibrium, and consequently no dyspnœa may remain.

If, however, bronchiectasis should occur in the compressed lung, the condition is much less favourable.

There may be more or less permanent displacement of the heart and other organs, obliteration of the intercostal spaces, overlapping of the ribs, and curvature of the spine when considerable retraction has occurred.

COMPLICATIONS OF ACUTE PLEURISY

The various exciting causes of the pleural inflammation mentioned under Etiology may be also regarded as complications, but they do not require detailed consideration here. Some degree of *bronchitis* is occasionally present, but cannot be considered a common complication. Pneumonia is invariably attended with an effusion of plastic lymph on some part of the visceral layer of the pleura corresponding to the site of the consolidation. It may also, although very rarely, occur in the opposite lung as a true complication of a primary pleurisy.

Pericarditis occurs in about 10 per cent. of the cases (Ziemssen); it is more often met with as a complication of purulent than of sero-fibrinous pleurisy, and is more common when the left side is affected than the right. It may be due to simultaneous infection of both serous membranes, or to direct extension through the lymphatics. The character of the pericardial effusion is generally similar to that of the fluid contained in the pleura. When, however, the effusion into the pleura is secondary to pericarditis with abundant purulent exudation, the fluid may be serous, and is then possibly due to pressure upon the azygos veins. Under such circumstances reaccumulation of the fluid in the pleura may repeatedly occur after its removal by paracentesis so long as the pericardial effusion remains.

Peritonitis is not of common occurrence, except in cases of empyema due to septic infection or of general tuberculosis of the serous membranes. In acute tubercular pleurisy there is often an infiltration of the peritoneum on the under surface of the diaphragm.

Meningitis is most likely to be met with in cases of pneumococcal infection; it is more common in children than adults, in whom it is an event of most rare occurrence.

Dilatation of the right cavities of the heart may take place in cases of long-standing effusion.

Acute endocarditis and embolism of the cerebral vessels is also occasionally met with in cases due to septic infection.

Multiple arthritis has also been observed under similar circumstances.

Gangrene of the pleura and lung is a rare complication of empyema. Necrosis is usually due to the action of septic organisms upon tissue which has been deprived of its blood supply, owing to compression or to thrombosis of the bronchial arteries. The change may, however, affect the parietal pleura and the adjacent intercostal muscles, but this condition is rare. A putrid empyema may be the result of primary gangrene of the lung.

Retraction of the chest wall usually occurs in chronic cases after a time with displacement of organs and some degree of compensatory enlargement, either of the uncompressed parts of the lung on the affected side, or of the opposite lung.

Calcification or ossification of the costal cartilages may also occur, and in rare cases a new growth of bone has been observed upon the cartilages and ribs leading to marked thickening and greatly increased rigidity.

Cerebral abscess is an occasional complication of chronic empyema. A well-defined abscess cavity containing greenish pus is usually present, but a diffuse softening has been observed, and in some cases has apparently been due to embolism of the cerebral vessels. The abscess may be single or multiple; the sites are variable, the temporo-sphenoidal, occipital, and parietal lobes being most often affected.

Purulent effusion unrecognised or operation delayed.—If the presence of a purulent effusion is not recognised or operation is unduly delayed various events may follow.

(1) **Perforation of the thoracic wall**, generally at one point, but occasionally at two or more, accompanied by denudation of the periosteum or perichondrium of the adjacent ribs or cartilages may occur. This may be due to direct ulceration of the pleura, or the formation of a peri-pleuritic abscess which may establish a communication with the sac before external rupture takes place.

The most common site of perforation is in front, in the fifth intercostal space just external to the pectoralis major (Marshall). The site of discharge may, however, be anywhere, not uncommon situations being the lower and posterior aspect of the chest, or even just above or below the clavicle.

After perforation of the parietal pleura the pus may burrow along the muscles, and may point in the lumbar region or appear below in the groin and simulate a psoas abscess. Rupture may also take place into the œsophagus or pericardium, or the diaphragm may be perforated and the pus be discharged into the stomach, or an abscess may form between the diaphragm and the liver. All the latter conditions, however, are of comparatively rare occurrence.

(2) **Perforation of the lung** may lead to the rapid discharge of a large quantity of pus through the bronchi, with the escape of air into the sac (pyo-pneumothorax). In some cases, however, particularly those of tubercular origin, more or less extensive superficial destruction of the lung occurs without the formation of a bronchial fistula. In other cases the opening is valvular and the discharge intermittent, or it may be cribriform and multiple, and the pus may reach the bronchi after passing through spongy lung (Traube), and under such circumstances pneumothorax does not necessarily ensue.

Perforation into a bronchus may occur after free drainage of the cavity has been established by operation, and also when spontaneous discharge has taken place. A bronchial fistula is rarely formed until an empyema has been in existence for a considerable period, generally from six weeks to two months. The condition resulting after absorption of the fluid parts of the effusion has taken place is described above.

Fortunately, nowadays, few cases are seen in which external rupture or even pointing has occurred. The condition usually implies that the disease has been present for a long period, and the lung may be incapable of re-expansion, owing to extreme thickening of the visceral layer of the pleura. The discharge of the pus, either spontaneously or by operation, may in such cases be followed by a long illness from failure of the sac to close by granulation. This may necessitate resection of portions of several ribs, but recovery may still be hoped for, although with a permanently damaged lung. After rupture into the lung, free drainage of the sac by operation may be followed by closure of the bronchial fistula and recovery. Before the days of paracentesis both external rupture and perforation of the lung were regarded as of favourable import, and cases are recorded in which even rapid recovery followed the escape of the pus.

THE CAUSES OF DEATH IN PLEURISY

Death may take place suddenly when the previous condition of the patient had not suggested that life was in danger; such a mode of termination of the disease is fortunately, however, an event very rarely observed. Death may also occur after long-continued dyspnœa, and in chronic cases of empyema from conditions induced by rupture of the sac, or from amyloid disease, the result of prolonged suppuration. In 32 cases analysed by Wilson Fox,[1] in which death occurred more or less suddenly, the mode was as follows:

---

[1] *Op. cit.* p. 1085.

	Cases
Suffocative or asphyxial	6
Syncopal (sudden)	20
Syncopal (gradual)	1
Combined syncopal and asphyxial	2
Doubtful	2
Doubtful syncopal (hæmoptysis)	1
	32

When death is preceded by long-continued dyspnœa ending in general collapse, there is generally œdema of the sound lung. The sudden discharge of an empyema into the bronchi may also be followed by death from asphyxia.

When death occurs from syncope in about half the cases some cardiac condition is present sufficient to account for the fatal termination. The following have been most often found: pericarditis; clots in the pulmonary artery, the result either of embolism or thrombosis; displacement, with compression of the large vessels, or displacement with obstruction of the inferior vena cava. In the remainder it is probably due to over-distension of the right cavities, followed by the formation of coagula. These changes are generally associated with failure of muscular power from a defective supply of oxygenated blood. Sudden death has in some cases been preceded by paroxysmal attacks of dyspnœa or by attacks of syncope.

Sudden death after paracentesis.—In 17 cases analysed by Wilson Fox, in which there was no doubt as to the cause, it was due to:

		Cases
Syncope	6	7
Probable syncope	1	
Asphyxia		5
Convulsions		4
Hæmorrhage from lung		1
		17

Fatal syncope may ensue from the withdrawal of even a moderate amount of fluid from the pleura, without any demonstrable organic lesion being present to account for death. The danger of this catastrophe is greater when the patient is beyond middle life, and is predisposed to by exhaustion, anæmia, and alcoholism, or by the assumption of the sitting position during or soon after the operation. It may also be simply the result of shock. In a limited number of cases it may result at a later period from thrombosis or from unexplained causes.

Death from asphyxia may be due to acute œdema of the lung accompanying its re-expansion; and such a condition may, by reflex vascular paralysis, extend to the opposite lung.

In all the cases recorded by Wilson Fox in which death was due to convulsions they occurred whilst the pleura was being washed out with fluid; but cases of this kind have been less frequently reported of late. It is probable that in some at least the fatal event has been due, in a manner which cannot be explained, to the fact that sufficient care was not taken to ensure that the fluid flowed out as fast as it entered. In some, however, a similar proceeding to that which ended fatally had been adopted several times without harm resulting, but in one case convulsions had occurred on a previous occasion whilst fluid was being injected. No lesion has been discovered to account for this mode of death.

The occurrence of some complication which may eventually prove fatal, such as rupture of the pus into the lung or elsewhere, is in many cases clearly due to an error in diagnosis.

DIAGNOSIS

Although in the majority of cases the diagnosis of pleurisy is unattended by any difficulty, it is probable that this disease passes unrecognised more frequently than almost any other occurring within the thorax. For this there is very often but little excuse, but the signs in some cases may be so misleading that the most experienced may fall into error.

In the early stage the absence of a friction sound is the chief cause of difficulty. Pleurodynia and intercostal neuralgia are unattended by pyrexia, and in those affections the tenderness is generally more marked than in pleurisy.

When a patient complains of pain in the chest he should always be directed to place one finger over the site of its greatest intensity. Careful examination should then be made along the whole course of the intercostal nerve or nerves apparently involved, and it should be borne in mind that the lower intercostal nerves are distributed to the muscles and skin of the upper half of the abdomen, and that consequently pain due to an inflamed pleura may be referred to the hypochondriac or epigastric regions.[1]

Pain referred to symmetrical points on the chest or upper half of the abdomen is generally due to spinal disease, but may be an indication of the presence of pleurisy on both sides. In some cases, from causes difficult to explain, pain may be referred to the side opposite to that on which a pleural friction is present.

Long-continued incomplete expansion of the side, from whatever cause arising, is, as already stated, followed by partial collapse of the lung, and on deep inspiration fine crackling râles may be produced by the expansion of collapsed alveoli upon the surface. This is differentiated from the crepitant friction sound of pleurisy by the fact that it occurs at the end of inspiration, whereas the pleural friction is usually audible immediately the chest begins to expand.

The diagnosis of pneumonia from acute pleurisy with effusion is fully discussed in the chapter on Pneumonia (*vide* p. 226).

We may, however, repeat that the *position of the cardiac impulse* is the most important point to be determined. If in a doubtful case it is much displaced, a pleural effusion is most probably present. In a case of double pleural effusion this valuable sign is wanting, but one of that nature in which vocal fremitus is also retained, and tubular breathing is present, is fortunately not likely to occur.

---

[1] *Vide* Hilton. *Lecture on Rest and Pain*: 3rd Edition, pp. 257, 252.

For the diagnosis of an effusion encysted above the diaphragm, and surrounded by spongy lung or in the interlobar fissure, exploratory punctures are usually necessary. The history of the case is important, as the evidence obtained from physical examination is likely to be inconclusive or misleading. The diagnosis from pleurisy of hydatid tumours of the lung and pleura and of mediastinal tumours is given in the chapters on those diseases.

The diagnosis of 'pneumonia with pleural effusion' is often made when there is really no inflammatory consolidation of the lung. In such cases there is usually absence of vocal fremitus, but a distant tubular breath sound is audible over the dull area. This is the glottic sound conducted through the still patent larger bronchi of the partially collapsed lung; it is generally most distinct towards the spine, but may, as already stated, be audible all over the dull area. Here again the position of the cardiac apex is the most trustworthy sign. When an inflammatory consolidation of the lung is associated with an acute pleural effusion, the resulting signs are a combination of those characteristic of each lesion, and their interpretation may not be free from difficulty. True crepitation may be present and the tubular breathing is not so distant as when the lung is compressed. The two conditions may not occupy exactly the same area, and at the margin undoubted signs of pneumonia may be present. Vocal fremitus may be weak, but not entirely absent, whilst the heart is displaced. The presence of rusty sputa is diagnostic of pneumonia under such circumstances. The coexistence of the two affections is in some cases indicated by the fact that all the physical signs become more marked the nearer one approaches the base of the lung, *i.e.* the percussion note becomes increasingly dull, the enfeebled vocal resonance is weaker or absent, and the breath sounds gradually disappear.

A large pericardial effusion is liable to be mistaken either for a left-sided pleural effusion or for pneumonia of the left lower lobe. The latter mistake depends upon the fact that, owing to compression and collapse of the base of the left lung, faint tubular breathing may be present over the posterior aspect of the lower lobe.

The position of the cardiac impulse is again the most trustworthy sign. If it is not found to be displaced to the right, a diagnosis of left-sided pleurisy should be made with much caution. The impulse of the displaced heart can, as a rule, be easily felt in pleurisy with effusion, whereas, when a large quantity of inflammatory exudation is present in the pericardium, the impulse is usually weak or absent, and the heart sounds are feeble and distant. In a case, however, of general dropsy from valvular disease in a child, we have observed the impulse of the heart to be well mar all over the greater part of the precordial area, although autopsy showed that the pericardium contained twenty ounces of serous fluid, and the lower lobe of the left lung was collapsed. The presence of orthopnœa and of the peculiar faint cyanotic tint of the face and extremities which are characteristic of pericarditis with much effusion, and the extension of the dulness upwards and its pear-shaped outline are signs and symptoms which should suggest the true nature of the case. Vocal fremitus is retained over the lower lobe when it is merely compressed, and the tubular breath sound is faint and distant.

The preceding remarks apply in part also to another condition in which greater difficulty in diagnosis is likely to arise, viz.: when a considerable effusion is simultaneously present in the left pleura and the pericardium, as under such circumstances neither pleural nor pericardial friction sounds are generally audible.

Very often the pleural effusion is recognised and the fluid is withdrawn, but the anticipated relief to the symptoms does not occur, and the cardiac impulse does not return to its normal position; it may, indeed, not be possible to localise it, and if this be the case suspicion should be aroused.

The diagnosis of a pleural effusion at the right base from various conditions of the peritoneum and abdominal viscera is considered on pages 477 and 478.

DIAGNOSIS OF THE NATURE OF THE FLUID

The nature of the fluid can only be absolutely determined by an exploratory puncture; there are, however, certain symptoms and signs which suggest, although they do not prove, that it is purulent. Exploratory puncture, if proper precautions are taken, is free from risk and is a method of diagnosis now so generally employed that a detailed discussion of this question is hardly necessary. In the accompanying table (p. 571) the clinical features of typical cases of purulent and sero-fibrinous pleurisy are stated, but it is important to remember that in some cases of empyema all or nearly all the differentiating signs here given may be wanting.

PROGNOSIS

The prognosis is chiefly influenced by the nature of the exciting cause, the age of the patient, the presence of complications and the amount and character of the effusion.

The nature of the exciting cause has a most important bearing on prognosis, as it, to a great extent, determines the character of the effusion and the occurrence of complications. The mortality of pleurisy associated with septicæmia, particularly in the puerperal state, and during an attack of scarlet fever, is relatively high, as the effusion is then usually purulent, and is often accompanied by pericarditis and peritonitis. Cases in which the effusion is putrid, the exciting cause being gangrene of the lung or pleura, are also extremely unfavourable. The influence of the various micro-organisms, which may be the immediate cause of the inflammation, has been already considered in discussing the etiology of the disease.

DIFFERENTIAL DIAGNOSIS OF PURULENT AND SERO-FIBRINOUS EFFUSION

—	Purulent	Sero-fibrinous
Age.	Common in children.	More common in adults.
Mode of origin.	Pneumococcus or streptococcus infection. Less common in tubercular pleurisy.	Common in tubercular pleurisy.
Mode of onset.	Early symptoms may be severe. 'Pneumonic' type of onset in pneumococcus infection.	Early symptoms may be severe.
Later symptoms.	Emaciation. Profuse perspiration. Repeated rigors. Febrile urine. Increasing anæmia, sallow complexion. Prostration } in adults. Delirium }	These symptoms are usually absent.
Character of pyrexia.	In early stages there is nothing typical. In later stages marked 'hectic' type is common. Pyrexia may be absent.	Fever declines in from 7–21 days as a rule. Crisis may occur. 'Hectic' type often observed in tubercular cases, and may persist for long periods.
Leucocytosis.	Present.	Generally absent.
Local changes.	Fluctuation in interspaces. Pointing. Pulsation. Œdema of chest wall. Rupture of sac.	Seldom observed. Very rare. Very rare. May occur, but is rare. May occur, but is rare.
Absorption.	Rare, but may occur. Occasionally observed in pneumococcus cases in children.	Common. Formed solid elements may long remain unabsorbed.

In sero-fibrinous pleurisy the complications most to be feared are double effusion and pericarditis. Pleurisy secondary to pneumonia and associated with extensive effusion is a grave condition, owing partly to the fact that the presence of the fluid is very likely to be overlooked.

The amount of the effusion certainly exercises some influence on prognosis, although absorption may be as rapid and recovery as complete with a large as with a moderate quantity. As a rule, recovery is likely to be less complete the longer the effusion is present.

In children and young adults, acute, primary, uncomplicated pleurisy, with sero-fibrinous exudation, rarely terminates fatally. In the great majority of such cases the effusion is absorbed, and recovery takes place within a few weeks, although the general health may remain impaired for a much longer period. The mortality is probably not much more than 2 per cent. In a few cases a more prolonged illness results, but even then a change to the chronic type is seldom observed. The probability of tubercle being present in many of the cases in young adults must be borne in mind in forecasting the future of the patient. With advanced age, when however serous effusion is not common, the prognosis is not so favourable.

Purulent pleurisy is characterised by a much higher rate of mortality, but this is gradually being reduced by earlier diagnosis and improved methods of treatment. In children the prognosis is as a rule favourable; this is shown by the fact that of fifty cases referred to by Dr. Goodhart[1] recovery was complete in forty-two, whilst in three a sinus remained, and only five ended fatally. Of the fatal cases, one was complicated by suppurative pericarditis, in one the empyema was double, another was due to the presence of a foreign body in the bronchus and septic pneumonia; in a fourth the disease had lasted eighteen months and had probably been overlooked, as a large collection of pus was present on admission to hospital; the fifth case, whilst progressing favourably, acquired measles and died from broncho-pneumonia. The causes of death in these cases, with the exception of the one last mentioned, which may be excluded from consideration, illustrate some of the conditions which influence the prognosis in empyema. This, indeed, greatly depends upon two factors—early recognition and the immediate adoption of proper surgical treatment.

TREATMENT

It is hardly necessary to state that a patient with acute pleurisy must rest in bed, but it may be well to point out that if effusion occurs he should be kept there until absorption, at any rate of the fluid part of the exudation, is completed.

The first indication is to relieve the pain in the side. If this is very severe, four or six leeches may be applied over the spot, unless the age or general condition of the patient renders their u-advisable. In children or old people and in weakly subjects they should not be employed. Hot linseed meal poultices or hot fomentations sprinkled with laudanum, frequently renewed, usually give great relief and are preferred by most patients to an ice-bag, although we have seen intense pain disappear quickly after the application of cold.

---

[1] *Diseases of Children*, p. 368.

A hypodermic injection of morphia may be given if the pain is not quickly relieved by local applications. In diaphragmatic pleurisy the pain and general distress are often severe and the use of morphia is more frequently necessary than in ordinary cases.

The application of blisters in this stage is rarely advisable, but the objection does not apply to the use of a mustard leaf or a mustard and linseed poultice. Leeches are generally more efficacious than cupping in the relief of pain.

If cough is severe and aggravates the pain, a linctus containing morphia or codeia may be given, unless, as is rarely the case, extensive bronchitis is also present. The administration of antipyretic drugs, solely with the object of reducing the temperature, is rarely advisable and usually harmful. If there is high fever it is, as a rule, better to control it by sponging the surface of the body for from ten to fifteen minutes at a time, with water at a temperature of 80° F. This may be repeated as often as the condition returns. In cases of a mild type, when a slight chill is followed by a stitch, rather than intense pain in the side, a broad band of strapping applied from the spine to the sternum may suffice to give relief by limiting the movement of the affected side. If with this a mixture containing fifteen grains of bicarbonate of potassium and of salicylate of sodium is given every three hours, until four or six doses have been taken, sweating is often produced and the fever disappears. These are, however, cases of localised inflammation with fibrinous exudation, and a similar result is not to be anticipated when the onset of the attack is accompanied by severe symptoms. In cases of a more severe type it is well to give at first a saline mixture containing citrate of potassium and acetate of ammonium, in order to promote the action of the skin and kidneys. The bowels should be freely opened, saline purgatives such as sulphate of magnesium or sodium being the most suitable agents for this purpose. At a later period, iodide of potassium (gr. iij) or squills may be added to a saline mixture.

The amount of effusion being moderate, this treatment may be continued for ten days or a fortnight; if then, or at an earlier period, the temperature falls, and the physical signs indicate that absorption is in progress, syrup of the iodide of iron may be given in combination with the iodide of potassium. A dry diet, combined with the administration of brisk saline cathartics and diuretics, is believed by some writers to promote the absorption of an effusion. Counter-irritation over the site of the effusion is useful at this stage; equal parts of the tincture and liniment of iodine form an application of a strength suitable to most cases, but in some patients the skin is unusually susceptible to the effects of iodine, and after it has been painted a few times a severe blister may be produced. The use of small flying blisters is preferred by some physicians.

After the fluid portion of the exudation has been absorbed dulness may remain at the base for some time, owing to the presence of coagulated lymph and partly to the incomplete expansion of the lung; if so, the local application should be continued. If fever is absent the patient may now be allowed to get up, confinement to bed at this period being harmful, as it tends to limit the free expansion of the lower part of the chest. Great care is necessary during convalescence, more particularly now that the possible presence of tubercle in many cases must be admitted, and so long as any morbid signs remain the patient should be kept under medical supervision, and not be encouraged to think that he has recovered.

The treatment of acute pleurisy up to the point thus far considered presents no difficulty, and gives rise to but little difference of opinion. When, however, after an illness lasting from ten days to a fortnight, a considerable effusion is present and the pyrexia continues, the question of paracentesis arises; and it has not proved easy to establish rules for its performance which are either generally accepted or applicable to all cases.

In the absence of any urgent symptoms and of the signs of positive intra-thoracic pressure it is usually better to defer operation for a few days longer, as there is but little danger in a case of sero-fibrinous effusion of this duration that the lung will not re-expand if absorption occurs, and the probability of the fluid reaccumulating after paracentesis is certainly greater if the operation is performed during the continuance of pyrexia. In the experience of the writer the unexpected absorption of an effusion is an event of far more frequent occurrence than its persistence when cure by the natural process appeared probable.

If, however, there is still no sign after some days that absorption is taking place, or if—the fever having disappeared—a considerable effusion remains, it is generally better to withdraw as much of the fluid as will flow through a syphon aspirator.

It has frequently been observed that the withdrawal of a comparatively small quantity of fluid is followed by the absorption of the remainder; this is probably due to the fact that the lymph channels are opened up by the removal of pressure.

Indications for paracentesis.—Under the following conditions paracentesis should be performed, irrespective of the duration of the illness or any other circumstance:

1. When there are signs of positive intrathoracic pressure.

2. When the following symptoms which usually accompany the above condition are present—a small irregular pulse and urgent dyspnœa, palpitation on slight exertion, lividity, or evidence of engorgement and œdema of the opposite lung. These symptoms may, however, be absent in cases accompanied by positive intrathoracic pressure so long as the patient is lying perfectly still.

3. When the fluid has been ascertained to be purulent, its removal is necessary in all acute cases (*vide*, however, Pyopneumothorax, p. 642).

If the purulent nature of the effusion is first discovered during the operation of paracentesis, no harm will be done by withdrawing as much of the fluid as will flow through a syphon aspirator; as, whilst the fluid continues to escape, the sac of the empyema must be diminishing in size, either through the expansion of the lung or by the approximation of the walls of the cavity; and in either case the subsequent obliteration of the sac, after the performance of the ordinary operation for empyema, is facilitated. If, however, the effusion is putrid, the operation should be performed without any delay.

The treatment of cases of recurrent effusion, and of those in which incomplete absorption with retraction occurs, is considered under Tubercular Pleurisy (*vide* p. 589), as many of them are of that nature.

J. K. F.

CHAPTER LI

CHRONIC PLEURISY

PAGE
Chronic pleurisy with effusion 576
Latent pleurisy 576
Chronic dry pleurisy 577
Mode of formation of thickened pleura with retracting lesions . 578

Two forms of this affection are recognised. In the one there is effusion present; in the other the pleurisy is 'dry.'

Chronic pleurisy with effusion.—Cases of this kind are, we think, less commonly met with at the present time than formerly. In some, the passage from the acute to the chronic stage occurs whilst the patient is under observation. Paracentesis is performed, but the fluid reaccumulates; the operation is then repeated with a like result; and this sequence of events may recur until the chest has been emptied and has refilled half a dozen times or more, the fluid continuing throughout of a serous character. It is then recognised that it is better to leave the case alone so far as active treatment is concerned. The fluid probably remains unabsorbed for a long time, but gradually some degree of retraction of the side takes place, indicating that the quantity has diminished. It may, however, remain for years unchanged, and, beyond some dyspnœa on exertion, the patient may suffer from few or no symptoms, and may be able to follow his occupation if it is not very laborious. In some of these cases at least it is highly probable that the pleurisy is tubercular.

'Latent' pleurisy.—Chronic pleurisy with serous effusion may be of the variety known as 'latent.' The onset of the attack may not have been characterised by any very marked symptoms—not, at any rate, by any so urgent as in the opinion of the patient to require medical advice—or the acute symptoms may have been of short duration, the early occurrence of effusion giving relief from pain.

As the fluid accumulates dyspnœa appears, and the patient finds that he is no longer able to continue at work, and on examination a large effusion is discovered.

Close inquiry into the history of these cases generally proves that the illness has not been quite so free from symptoms as would appear from the first statement of the patient. The patient is, however, generally unaware of the presence of any serious disease, and pain and fever are absent.

'Latent' peritonitis has of late been proved by the frequent performance of laparotomy to be tubercular, and, if it were possible to see the pleura, it is highly probable that in many cases of latent pleurisy a like condition would be found.

Clubbing of the fingers and toes is often present in these cases, and may be extreme.

Paracentesis should be performed, and if, as is usually the case, the fluid is found to be still serous, as much of it should be withdrawn as will flow through the aspirator. If it should reaccumulate a second aspiration is advisable. In nearly all cases of this kind repeated tapping is practised, and no definite statement is possible as to the exact number of times it is advisable to operate. But sooner or later the opinion is formed that no further attempts should be made to remove the fluid. Active counter-irritation may then be continued, and the treatment directed to the improvement of the general health. A residence for a time at a high altitude may be tried in suitable cases, and is often followed by recovery with re-expansion of the lung.

If the fluid proves to be purulent, the ordinary operation for empyema should be performed at once, unless there is good reason to believe that the case was originally one of pneumothorax, secondary to pulmonary tuberculosis (*vide* p. 596).

Chronic dry pleurisy.—Pleural adhesions are very commonly found on post-mortem examination when the clinical history contains no record of a previous attack of acute pleurisy. It is indeed rare to find them entirely absent in adults, unless the lungs are in a condition of general emphysema. The surfaces may be completely united on both sides or on one only; more often the adhesions are limited to certain areas.

Very extensive union of the pleural surfaces may be present, though the individual is unconscious of any impairment of breathing power; and, besides those adhesions which result from acute plastic or sero-fibrinous pleurisies, it is probable that others may be produced by inflammatory processes of a subacute character, which are unaccompanied by obvious symptoms.

Fine filamentous adhesions may be unattended by any distinct signs, but any decided thickening of the pleura is usually accompanied by some degree of retraction with inspiratory recession of the interspaces.

When there is complete union over one side, the signs present depend to a great extent upon the nature of the adhesions and the degree of thickening of the pleura. The expansion in such cases is generally diminished, and inspiration may end abruptly; the breathing may be vesicular in quality, but weak. Vocal fremitus is generally diminished, but not abolished.

Surgical proceedings have proved that it is practically impossible to be certain whether or no the surfaces of the pleura are adherent. In the course of operations undertaken for the drainage of cavities in the lung, after confident opinions had been expressed by experienced physicians that the pleural surfaces would be found adherent, they have proved not to be so, and it has been necessary to stitch them together and wait for union to take place before proceeding to open the cavity.

The late Sir Andrew Clark[1] held very strongly that a non-tubercular dry pleurisy might lead to great thickening of the membrane, and that the inflammatory process might invade the lung and induce fibroid changes in it, a condition to which the term 'pleurogenic cirrhosis' is now generally applied. We are disposed to doubt whether this condition of the lung is often produced in such a manner, but great thickening of the pleura and pulmonary fibrosis are certainly frequently found in association.

In fig. 78, taken from a case of subacute pleurisy, the thickening of the interlobular septa is shown, and such a condition is not infrequently observed in chronic pneumonia apart from tubercle (*vide* p. 265).

Chronic adhesive tubercular pleurisy, of which great thickening of the membrane is the most marked feature, is described elsewhere (*vide* p. 586).

In cases of very chronic pulmonary tuberculosis (the fibroid phthisis of Andrew Clark) the upper lobe or the whole lung may be surrounded by a fibrous covering of almost cartilaginous density. The various stages in the formation of this lesion are as follows: Local disease in the lung leads to the formation of adhesions between the surfaces of the overlying pleura; the lung, carrying with it the visceral pleura retracts in consequence of the fibrous changes taking place within it; the adhesions are stretched, and fine vessels can be seen within the fibrous bands, and between them yellow serous fluid is effused, which at this stage can be easily squeezed out after section.

As the lung continues to retract the surfaces of the pleura are still farther separated, and at a later period the exudation becomes paler in colour and gelatinous in consistence, and the fluid can no longer be expressed. Organisation of the effusion is now in progress, and it is gradually transformed into fibrous tissue, which in process of time acquires the density of indiarubber and later of cartilage. A lung thus enveloped can only be removed from the chest with a sharp knife.

If the lung is seen before this extreme stage is reached, it is found to be marked with lines of yellow fat, which correspond to the ribs and not, as would appear at first sight, to the interspaces. The resistance of the ribs to the retracting force exercised by the fibrous tissue in the diseased lung being greater than that of the interspaces, fat is deposited beneath the former in order to fill up the space which would otherwise be left, and this is at a subsequent period replaced by fibrous tissue.

---

[1] Vide *Lumleian Lectures*, Royal Coll. Phys. 1885.

Whenever a retracting lesion is present in the lung, and is situated sufficiently near to the surface, one or other of two events always happens—either the pleura becomes thickened in the manner just described, or emphysematous bullæ are formed on the surface of the lung. If there is no lung tissue between the retracting lesion and the pleura capable of undergoing distension, the former change occurs; if there is, the latter lesion is produced.

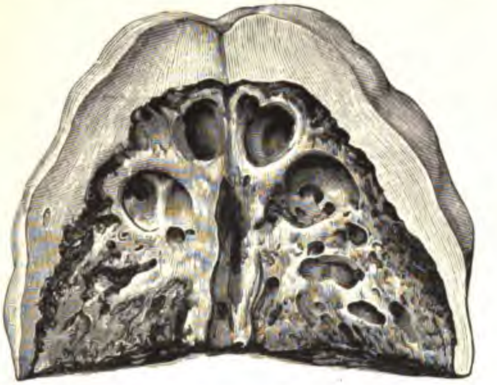


FIG. 132.—SHOWING THICKENING OF THE PLEURA SECONDARY TO CHRONIC TUBERCULOSIS OF THE LUNG

This, and not a proliferative change in the serous membrane, we believe to be the mode of production of the extreme thickening of the pleura observed in chronic cases of pulmonary tuberculosis, and in the majority of cases of non-tubercular cirrhosis of the lung.

J. K. F.

CHAPTER LII

TUBERCULAR PLEURISY

PAGE
Etiology 580
Clinical types 580
  1. Acute tubercular pleurisy 581
    (*a*) With acute onset 581
    (*b*) Terminal 582
    (*c*) Purulent 584
  2. Subacute and chronic 584
    (*a*) With sero-fibrinous effusion 584
    (*b*) With purulent effusion 585
    (*c*) Chronic adhesive 586
  3. General serous-membrane tuberculosis 587
Diagnosis 588
Treatment 589
APPENDIX: A brief account of the various forms of pleurisy from a bacteriological point of view 590

Etiology.—The visceral layer of the pleura is generally infected either through the sub-pleural lymphatics or from the bronchial or tracheal lymph glands. The virus may reach the parietal layer from the cervical, vertebral, or mediastinal glands, or through the diaphragm from the lymphatics of the peritoneum.

The lung is no doubt the most common source of infection, but the pleurisy which accompanies pulmonary tuberculosis and leads to the formation of the adhesions which are rarely absent in that disease is, in only a small proportion of cases, proved post-mortem to have been tubercular. It may be of that nature, but if the presence of miliary or caseous tubercles in the pleura is required in proof of the fact, the evidence is defective in the great majority of cases. In many cases it is certainly impossible to demonstrate their presence, and the pleurisy may arise from infection by associated organisms, which gain access from areas of inflammation near the surface of the lung. We do not, however, regard this question as decided, and are of opinion that if more careful search were made, especially in the interlobar septa (*vide* p. 544), the pleurisy could be proved to have been due to tubercular infection in a much larger proportion of cases than it is at present.

Clinical types.—In considering the etiology of pleurisy the question of the possible tubercular origin of cases occurring in apparently healthy individuals free from inherited tendency to tuberculosis, and marked by an acute onset with severe pain in the side, pyrexia, cough, and occasionally rigors and attributed to a chill, has been discussed, and the opinion was expressed that a considerable proportion of such cases are of that nature. It is therefore unnecessary to consider in detail the symptoms and physical signs by which they are accompanied, as, if this view is correct, they must obviously be to a great extent similar to those already described in the chapter on Acute Pleurisy.

In addition to the type of the disease, in which the exudation is usually sero-fibrinous but occasionally purulent, there are others marked by a subacute or insidious mode of onset and a chronic course, the exudation being either fibrinous and subsequently either gelatinous, sago-like or caseous, or presenting the characters just mentioned.

They have been most ably differentiated by Osler,[1] and we are indebted to him for the classification here adopted:

1. Acute tubercular pleurisy.
2. Subacute and chronic tubercular pleurisy.
3. General serous membrane tuberculosis.

1. ACUTE TUBERCULAR PLEURISY

This may be subdivided into

(*a*) Cases in which the onset is acute, but the subsequent course of the disease chronic.

The following case is described by Lemoine:[2]

A soldier was admitted to hospital January 28, 1892. He had lost a brother from pulmonary tuberculosis. The patient had never been ill, and presented all the appearances of an exceptionally vigorous constitution.

On January 16 he was seized with moderately severe pain in the right side, and complained of cough and loss of appetite. He continued on duty until January 27, with alternations of improvement and relapse.

On admission a large effusion was present on the right side. Aspiration was performed, and a litre of clear fluid withdrawn. Pyrexia continued up to March 18, the temperature ranging between 100·2° and 103°. From that date to April 2, fever was absent, but there was a marked difference between the morning and evening temperatures. Appetite returned, he was allowed to get up, and appeared on the way to recovery, although signs of effusion remained. He died suddenly on April 7 on rising after breakfast.

On post-mortem examination 500 grammes of sero-fibrinous exudation were found in the right pleura. The whole surface of the membrane was covered with miliary granulations, and at the apex of the cavity there were some caseous nodules the size of peas. Miliary tubercles were also present in the lung, and two bronchial glands were caseous.

---

[1] *Shattuck Lecture on Tuberculous Pleurisy*, 1893.

[2] *Bullet. des Hôp. de Paris*, 1895, p. 256.

In this case the fatal termination from syncope may be regarded almost as an accident. Had the patient lived it is extremely probable that the disease would have followed the chronic course often observed in such cases, viz.: repeated recurrence after tapping, followed eventually by recovery, and subsequently by the development or extension of pulmonary tuberculosis. It will be observed that a large quantity of fluid was present in the pleural cavity, and that the lung was already infected.

(*b*) **Terminal acute tubercular pleurisy.**—The affection may occur at the end of a variety of chronic diseases, such as chronic endocarditis, cirrhosis of the liver, chronic Bright's disease, general arterio-sclerosis, and others.

In some of these cases obsolete tubercle is present in the lungs, in others the infection of the serous membrane is primary. The true nature of the condition is rarely recognised during life. The exudation may be fibrinous, sero-fibrinous, hæmorrhagic, or caseous.

The following cases illustrate this type of tubercular pleurisy:

Case II.—Mary O., æt. 36, admitted to Brompton Hospital October 23, 1883; died July 11, 1884.

Family history of tuberculosis on the father's side.

Patient had had a cough in the winter for eight years.

Four years ago she suffered from a severe attack of acute rheumatism. The cardiac affection dated from this period. Cough had been worse for the last six months, and of late the patient had noticed œdema of the face and ankles, and the urine had been scanty.

On admission there were signs of great enlargement of the heart, and of regurgitation at both mitral and tricuspid orifices; there was also ascites, and the liver was enlarged. The urine contained $\frac{1}{4}$ albumen. During her stay in hospital paracentesis abdominis was performed more than twenty times.

Autopsy.—General firm adhesion of surfaces of pericardium. Heart greatly enlarged ($18\frac{1}{2}$ oz.). Mitral and tricuspid orifices much dilated. Fibroid patch on inner wall of left ventricle, antemortem clot adherent to it: atheromatous patches on mitral valve. Four quarts of pale yellow serum in peritoneal cavity. No tubercle present. Liver (4 lbs.) typically 'nutmeg;' kidneys granular.

Both lungs adherent to pericardium. Left pleura contained about one pint of clear serous fluid. Recent lymph on pleura and miliary tubercles, also on the parietal pericardium between the left lung and the heart. Two or three firm caseous nodules in each lung, with few recent miliary tubercles around.

Bronchial and mediastinal glands contained caseous nodules and miliary tubercles.

Case III.—Norah M., æt. 18, admitted into the Brompton Hospital November 28, 1893; died March 13, 1894.

No history of tubercular disease in the family. In 1891 she had rheumatic fever, followed by a relapse. She has had a cough in the winter for an indefinite period. Health has gradually failed for the last ten weeks. Five weeks before admission she had hæmoptysis (one teaspoonful). No adventitious sounds were audible in the lungs on admission, but the breath sounds were bronchial at the right apex. Crepitant râles subsequently appeared in the right supra-spinous fossa. The temperature was normal or sub-normal until February 25, when continuous pyrexia developed (100°–102° F.), and signs of meningitis.

Autopsy.—Heart: left ventricle hypertrophied, right dilated; marked stenosis of mitral orifice, and great thickening of both flaps of the valve and of the chordæ tendinæ; the latter matted together at their insertions. Left auricle dilated. Aortic cusps thickened.

Right pleura: visceral layer covering right upper and middle lobes densely infiltrated with miliary granulations, which were also present in the interlobar fissures. About ten ounces of clear fluid in cavity. Patches and flakes of lymph on pleura, chiefly over site of tubercular infiltration. No adhesions.

Miliary tubercles in right upper lobe and a small caseous focus undergoing softening; none in left lung.

Basic meningitis, probably tubercular. Tubercular ulcer at lower end of ileum.

Case IV.—Henrietta B., æt. 36, admitted into Brompton Hospital October 22, 1884; died December 26, 1884.

Family history of rheumatism, none of tuberculosis. Patient has twice had acute rheumatism, the last time three years ago.

For the last year has suffered from cough, which has been worse for the three months preceding admission. Dyspnœa for two months, and pain in the right side. Three months ago had hæmoptysis; the blood looked like 'lumps of flesh.'

On October 28, paracentesis; 3½ pints of serous fluid withdrawn from right chest. On the following day the patient had a rigor and sweated profusely, and the evening temperature rose to 101°. Pyrexia was subsequently continuous.

On November 14 a free incision was made in the right pleura between eighth and ninth ribs in axilla, and 5½ pints of fœtid pus evacuated. The temperature then fell suddenly to 97·8.

On December 16 the purulent discharge was found to contain 'myriads' of tubercle bacilli. Thrombosis of the veins of the lower extremity was then present.

Autopsy.—Heart (8 oz.) small. Aortic cusps greatly thickened and puckered, but competent to water test; mitral valve thickened; ventricular walls thin.

Right pleura symmetrically and universally thickened, lined throughout with thick greyish yellow layer of pus; no fluid in cavity.

Interlobar septa thickened, and contained softening caseous material which communicated through a small opening with the cavity of the pleura; no appearance of an old pneumothorax. No communication with subjacent lung, which appeared to be healthy.

Right lung contained patches of pigmented fibroid induration enclosing caseous nodules, some of which were softening in the centre, others dry and mortary. Similar nodules and some recent miliary tubercles in the left lung.

(*Note by Dr. Percy Kidd.*—'It seems possible that after the first puncture of the chest the caseous matter in the interlobar septum softened and effected a communication with the pleural cavity. There may, on the other hand, have been a communication with the lung, which subsequently closed up.')

(*c*) **Acute purulent tubercular pleurisy.**—Purulent pleurisy of tubercular origin is rarely met with. The disease is usually insidious in onset and chronic in its course, but cases occasionally occur in which the illness commences with a rigor and severe pain in the side, runs an acute course, and is characterised throughout by severe symptoms. In one of the kind, described by Osler, which terminated fatally in so short a period as five weeks, extensive ulcerations were present throughout the right pleura with pockets of pus between firm adhesions, the membrane was generally thickened, greyish-white in colour, and extensively infiltrated. There was an acute miliary tuberculosis of both lungs and of the left pleura.

2. SUBACUTE AND CHRONIC TUBERCULAR PLEURISY

(*a*) **With sero-fibrinous effusion.**—Although, as already stated, the onset may be, and often is, acute in cases of tubercular pleurisy with sero-fibrinous effusion, many cases are characterised by an insidious mode of invasion and run a chronic course. The nature of the disease may in some be inferred, either from previous symptoms pointing to the presence of pulmonary tuberculosis, or from the constant reaccumulation of the fluid after paracentesis. A fatal termination is often preceded by an acute illness due to a rapid dissemination of tubercle. In others, after partial recovery, with absorption of the fluid portion of the effusion, death occurs at a later period from pulmonary tuberculosis.

In a considerable number of cases of the latter disease the pleura becomes infected at some period of the illness, and a sero-fibrinous effusion forms; but in those here referred to the pleural lesions are, if not primary, at any rate for a time more prominent, and, when death occurs, may prove of older date than the changes in the lungs.

The following case is an example of subacute tubercular pleurisy with sero-fibrinous exudation.

Case V.—Blanche R., æt. 19, admitted to Brompton Hospital March 16; died May 28, 1894.

There was no history of tubercular disease in the family.

The present illness began a month before admission, with severe pain in the right side and cough; the latter symptom has continued. There has been no hæmoptysis emaciation, or dyspnœa. On admission there were signs of the presence of a considerable effusion in the right pleura. Paracentesis was performed on March 30, and 62 oz. of serous fluid were withdrawn. The operation was repeated on May 18, and 18 oz. of blood-stained serum removed. Death occurred suddenly on May 28 from cardiac failure.

Autopsy.—The left pleura contained 20 oz. of serous fluid; the membrane was studded with miliary tubercles, in places aggregated into large patches.

Costal and parietal layers of right pleura greatly thickened and covered with abundant irregular masses of caseous tubercle; the two layers were adherent as far as the third rib; from that point a longitudinal partition of lymph completely divided the cavity into two separate compartments; it was attached below to the diaphragm. A few areas of recent yellowish-grey tubercular pneumonia were present in both lungs, but were clearly of later date than the tubercle on the pleura. A few tubercles were found on the surface of the pericardium and also of the peritoneum.

The following case is an example of infection of the pleura occurring in the course of an ordinary case of chronic pulmonary tuberculosis.

Case VI.—Robert M., æt. 30, brickmaker, admitted into the Brompton Hospital August 5, 1892; died September 3, 1892.

No family history of tuberculosis. Patient had had no serious illness, but had been 'languid' since he was twenty-five years old. For the last twelve months he had suffered from cough and expectoration; had never had hæmoptysis; no diarrhœa. After admission there was continuous pyrexia, and the physical signs indicated a rapid extension of the disease.

Autopsy.—The visceral and parietal layers of the right pleura were infiltrated with miliary tubercles; they were especially numerous over the lower lobe. There was no lymph either surrounding them or in their neighbourhood. There were firm adhesions over the left pleura.

Both lungs were extensively excavated and contained caseating tubercles. The larynx and intestines showed tubercular ulceration.

(*b*) **With purulent effusion.**—Cases of tubercular pleurisy of the subacute or chronic type, in which the effusion is purulent, are of very rare occurrence, apart from pneumothorax. Possibly in some the true nature of the disease is overlooked, as most careful microscopical research is necessary in order to determine the presence of tubercle in the thickened lining of a chronic empyema.

The following case is interesting from the fact that the pleurisy, although tubercular, appeared to be connected with a fracture of the ribs.

Case VII.—A man, æt. 70, was admitted into the Middlesex Hospital on May 14, 1889, and died June 15 following.

Some months before his death he sustained an injury to the right side which resulted in a fracture of the seventh and eighth ribs.

Autopsy.—Fifty ounces of turbid fluid, in which many flakes of lymph were floating, were present in the right pleura. The parietal layer posteriorly and inferiorly was thickly studded with tubercle. There were no tubercular deposits in the lungs. The kidneys were granular, and there was general arterio-sclerosis with hypertrophy of the left ventricle.

(*c*) **Chronic adhesive tubercular pleurisy.**—Slowly spreading tubercular infection of the pleura often leads to great thickening of the membrane, and this constitutes the distinguishing lesion in cases of this type. In some, however, of dry pleurisy, which are probably but not certainly tubercular, adhesion occurs without well-marked signs of thickening of the membrane either at the time or subsequently.

The writer has watched cases of dry pleurisy in tuberculous subjects in which the affection of the pleura could be traced upwards day by day, from one interspace to another, until finally the friction sound was audible over the actual summit of the lung ; subsequently it disappeared, probably owing to adhesion having taken place. Pain was absent or trifling, and there was but little fever or other signs of constitutional disturbance.

Although strongly disposed to think that a dry pleurisy of this type is generally tubercular, the absence of positive post-mortem proof and the admitted difficulty in demonstrating tubercles in the pleural adhesions of pulmonary tuberculosis render a decided statement on the question impossible.

Although primarily most marked in the pleura the change is often not limited to it, the other serous membranes being also involved, and some cases are hardly distinguishable post-mortem from those of the group following.

In some cases extreme thickening takes place without any effusion; in others a small amount of either sero-fibrinous or curdy or cheesy exudation is found, or marked thickening may be present over the upper part of the pleural sac, with effusion at the base.

Obsolete tubercular lesions are often present in the lungs, and in some cases more active tubercular lesions are found. Chronic interstitial lesions, chiefly due to thickening of the interlobular septa, may be set up in the compressed lung.

The following is a typical case.

Case VIII.—H. A., æt. 55, a mason, admitted to Brompton Hospital May 25, 1893 ; died June 24, 1893.

No family history of tuberculosis. No illness until winter of 1891–2, when he had an attack of bronchitis ; recovery followed, and his health continued good up to November 1892. Since then he had suffered from shortness of breath. On December 10 he had a severe illness, of sudden onset, beginning with pain in the right side. On admission, thin and weak. Signs of fluid (or growth ?) in right side, but no displacement of the cardiac apex.

Right chest aspirated, 6 oz. of blood-stained fluid withdrawn. Temperature irregular, but usually normal. Rose to 102° F. a week before death, which was preceded by urgent dyspnœa.

Autopsy.—Right pleura adherent, except at base, and much thickened, dense and fibrous, especially over diaphragmatic surface, where it measured from ½ to 1 inch between the two layers. At the base there was an effusion of 15 oz. of blood-stained fluid. Cavity lined with gelatinous material. Where thickening was least, small yellow caseous nodules present. Adjacent parietal layer of pericardium and under-surface of diaphragm dotted with tubercles. The diaphragm was as hard as a board. Bronchial and mediastinal glands infiltrated with tubercle. Old fibrous tubercles in both lungs.

3. GENERAL SEROUS-MEMBRANE TUBERCULOSIS

An acute miliary infection confined to the large serous sacs is by no means uncommon, the pleura and peritoneum being most often associated, while the pericardium may escape.

In more chronic cases great thickening of the pleura, and to a less extent of the peritoneum, occurs, or thickening of the serous membranes may be present with fibroid transformation of tubercle.

The writer had lately under his care at the Brompton Hospital a case of general serous-membrane tuberculosis, in which the pericardium was primarily involved; this was followed after some weeks by double pleurisy without effusion, and subsequently by infiltration and softening at the left apex; tubercle bacilli then appeared in the sputa.

The following cases illustrate the lesions found, and the symptoms and course which characterise cases of this type.

Case IX.—Georgina M., æt. 22, admitted to the Brompton Hospital January 27, 1890; died April 29.

Father and mother both died of 'congestion of the lungs.' The patient had pleurisy two years ago, for which she did not lie up. Cough has been present ever since. On admission the physical signs suggested a diagnosis of double pleural effusion. On February 15 paracentesis was performed on the right side, but no fluid was withdrawn. February 17 jaundice appeared. On February 27, 59 oz. of greenish-coloured serous fluid were removed from the left pleura. March 5.—Right pleura aspirated, but no fluid obtained. March 17.—Left pleura aspirated, and 17 oz. of bile-stained fluid withdrawn. Jaundice more marked. March 29.—Left pleura aspirated; 12 oz. of opaque bile-stained fluid withdrawn.

Autopsy.—Tuberculosis of pleura, pericardium, and peritoneum. Old circumscribed tuberculosis of lung. Caseous bronchial and mediastinal glands.

Both pleuræ adherent. The left occupied by a large empyema with thickened velvety lining on parietal surface; the visceral layer was thick, shaggy, and honeycombed; a caseous nodule was present at base; lung completely collapsed; fibrous tubercle in both upper lobes. Right pleura densely adherent; great thickening at base, with deposit of lymph and tubercle. Pericardium everywhere adherent, much thickened from tubercular deposit.

Caseous foci in bronchial and mediastinal glands.

Peritoneum studded throughout with miliary tubercles, single and in groups, most abundant in Douglas's pouch and pelvis, and about the liver.

Miliary tubercles in liver and kidneys.

Case X.—Henry J. S., æt. 15, admitted to Brompton Hospital March 19, 1888; died June 10.

No history of tuberculosis in family. Present illness began in January 1887, with pain in the left side. Cough and expectoration had been present throughout. Enlargement of abdomen first noticed two months before admission. On admission there were signs of a large effusion in the left pleura and also of ascites. Paracentesis was performed on March 23, and 10 oz. of clear serum withdrawn from the left pleura. March 28, operation repeated and 15 oz. of fluid removed. The urine gradually diminished in quantity, and death was preceded by coma.

Autopsy.—Tuberculosis of both pleuræ, pericardium, peritoneum, cerebral meninges, lungs, glands, &c.; no amyloid disease.

Large serous effusion in cavity of left pleura. Membrane greatly thickened from caseous infiltration. Opaque submiliary nodules in less caseous portions. Right pleura adherent; on separation, surfaces and exudation infiltrated with blood, and showing whitish nodules in groups; thick yellow area of caseation at the lower margin.

Left lung compressed, tough and airless, and containing greyish nodules. Grey granulations throughout right lung.

Surfaces of pericardium adherent and much thickened, infiltrated with caseating tracts and nodules.

General tuberculosis of peritoneum, but most marked on diaphragm.

Bronchial glands firm, dense and caseous, and probably primary seat of tuberculosis.

Mediastinal glands contain submiliary granulations.

Tuberculosis of spleen, liver, kidneys, and glands.

Diagnosis.—A careful perusal of the typical cases which have been recorded will show under what conditions the affection is likely to be met with; but, although the nature of the disease may be suspected, it is often extremely difficult to convert suspicion into proof.

The family history of the patient, his physique, the occurrence of previous symptoms pointing to pulmonary tuberculosis or the presence of enlarged glands may suggest a correct diagnosis; but a tubercular pleurisy may occur in an individual who has previously enjoyed good health and appears to possess a sound constitution. No reliance can be placed upon the mode of onset of the attack; but a very prolonged course, repeated recurrence of the effusion after paracentesis, and incomplete absorption followed by marked flattening, are conditions strongly suggestive of a tubercular origin. We have in several cases observed *absorption of the fluid during the continuance of pyrexia of a markedly remittent type*, and are disposed to regard this as an important indication of the tubercular nature of the disease. In all cases of pulmonary disease, no matter how apparently 'simple' in character, the sputum, if there is any, should be examined for tubercle bacilli, and it is especially necessary in cases of pleurisy. In the great majority of tubercular pleurisies the lungs at some period, either early or late, are involved in the process, and its true nature may thus be detected.

From what has been stated in the section on Etiology (*vide* p. 544) as to the results of the bacteriological examination of pleural effusions, it will be apparent that great assistance in the diagnosis of the cause of sero-fibrinous effusion is not to be expected from that method; but it should not be neglected when opportunity offers, and in purulent effusions a positive result is often obtained. In cases of general serous-membrane tuberculosis, in which the pericardium is primarily involved, effusion may occur to such an extent as to suggest the necessity for paracentesis, but temporary recovery may even then take place with complete absorption of the fluid. In most cases of this kind the general symptoms are far less severe than when the affection is of septic or even of rheumatic origin, and this fact alone may suggest a correct diagnosis. If, for example, a patient presenting signs of considerable pericardial effusion is able to lie flat in bed without dyspnœa, palpitation, or any sign of discomfort, and has only a moderate degree of pyrexia, and a fairly good appetite, while there is little or no evidence of constitutional disturbance, it is extremely probable that the disease is tubercular.

Treatment.—An attack of pleurisy is not under any circumstances an illness to be lightly thought of; but if there is reason to suspect that it is tubercular, more than ordinary care is necessary to obtain, if possible, the complete absorption of the exudation and re-expansion of the lung. As a correct diagnosis is rarely possible at an early period, the treatment must be similar to that suitable to non-tubercular cases. If the fluid, though still sero-fibrinous, repeatedly re-accumulates, it may be advisable, if fever is absent or moderate, to move the patient to more healthy surroundings where he can obtain the good effects which sunshine and a life in the open air exert upon the resisting power of the body; and this may be worth doing, even though most of the day is spent lying upon a couch.

In all cases of tuberculosis the maintenance of the nutrition of the body by an abundance of nourishing food is a matter of as much importance as the administration of drugs, but the latter may be required to improve the condition of the digestive organs. Good results have also recently been reported in cases of tubercular pleurisy from the administration of gradually increasing doses of creasote.

At a later period, if dulness remains at the base and retraction of the side is in progress, great benefit may follow a residence at a high altitude, for example, at St. Moritz, Davos, Arosa or elsewhere, both from the invigorating effect upon the body generally, and from the more complete expansion of partially collapsed lung which follows the breathing of rarefied air.

Various methods to which the term 'pulmonary gymnastics' is applied are in use for obtaining expansion of collapsed lung, such as forcing water from one large Wolff's bottle to another, and inspiration of compressed air by Waldenburg's, or Fraenkel's, or Ketchum's apparatus.

Opinions still differ as to the best treatment for cases of tubercular empyema unaccompanied by pneumothorax. If they are dealt with by incision and drainage, as some recommend, the closure of the cavity is very often incomplete, owing to the great thickening and rigidity of its walls, and in such cases a discharging sinus remains, and amyloid disease may follow, or death may be due to tuberculosis of the lungs. The subject is considered in the chapter on the Surgical Treatment of Empyema (*vide* p. 597).

J. K. F.

APPENDIX TO CHAPTER LII

A BRIEF ACCOUNT OF THE VARIOUS FORMS OF PLEURISY FROM A BACTERIOLOGICAL POINT OF VIEW

THE account of the various forms of pleurisy given in the preceding chapters may be usefully supplemented by a short description of the clinical characters which the disease acquires owing to the presence of the various micro-organisms mentioned. For the following description we are partly indebted to the observations of Straus, Fernet, and Lemoine.

Pneumococcus pleurisy (sero-fibrinous and purulent).

Etiology.—The affection of the pleura may be primary, but is more often secondary to pneumonia of a severe type, occasionally also to broncho-pneumonia and otitis. It may coincide with the onset of pneumonia, or may appear during its course; more often it occurs at the termination of the lung affection, sometimes several weeks later (Straus). Empyemata in children are generally of this nature.

Symptoms and course.—The mode of onset and the symptoms may exactly resemble those of pneumonia (Washbourn, Fernet). There may be a single severe rigor, high fever (temp. 102–104°F.), quick pulse (120–140), severe pain in the side, cough, short and dry, or with slight viscid or blood-stained expectoration. Labial herpes may be present. A crisis may occur about the seventh day, but relief is less complete than in pneumonia, as the dyspnœa persists. Straus, however, states that the pyrexia is generally moderate, and may be absent in purulent pleurisy of this nature.

Character of the exudation.—Sero-fibrinous effusions may be abundant or moderate in amount, yellow or greenish in colour, very fibrinous, and forming after withdrawal a large clot.

Purulent effusions are thick, greenish, creamy, not clotted. On standing the fluid does not readily separate into clot and serum, but a thin layer of transparent or greenish serum appears upon the surface after a time (Netter).

The exudation may at the beginning be sero-purulent in cases which afterwards become purulent. Soft false membranes easily detachable may line the two layers of the pleura; often they float in the fluid, but appear to be easily re-absorbed. The exudation is often partitioned off and encysted (Straus).

Prognosis.—In the sero-fibrinous effusion the duration may vary from some days to two or three weeks, rarely longer (Fernet). In purulent cases the prognosis is more favourable than in empyemata caused by or associated with the presence of any other micro-organisms. The usual period is from some weeks to two months (Straus). The affection rarely becomes chronic. A sero-fibrinous exudation may be completely re-absorbed, and this may also occur, but more rarely, after it has become purulent. If paracentesis is performed after the fifteenth day and the fluid is sero-fibrinous, the effusion rarely re-accumulates. The mortality is only from 10 to 15 per cent., in contrast with 25 per cent. in the other varieties. Convalescence may be hindered, and the prognosis rendered less favourable if streptococcus infection subsequently occurs.

Treatment.—Simple aspiration, even with purulent effusion, may be followed by recovery.

Empyemata of this nature, if left untreated, often terminate by spontaneous evacuation.

Purulent streptococcus pleurisy.

Etiology.—This is the most common variety of purulent pleurisy in adults. It may be primary, but is more often secondary to pneumonia, septic broncho-pneumonia, influenza, scarlet fever, throat affections, puerperal fever, and a variety of other diseases.

Symptoms.—The mode of onset may be acute, with severe pain, or it may be insidious. Pyrexia is often extreme and the temperature presents marked irregularity; but in some cases there is only a moderate degree of fever. Rigors may occur repeatedly.

Streptococcus infection may be the cause of purulent pleurisy following pneumothorax, due to the perforation of the pleura by a softening tubercular mass in the lung. A case of this kind has recently been under the care of the writer in the Middlesex Hospital.

Character of the effusion.—The effusion may be at first serous and the organisms may be found before the onset of suppuration; subsequently it becomes sero-purulent, and finally purulent. It is usually moderate in quantity, yellowish in colour, and not very thick. It separates, on standing, into two layers, of which the upper is clear and abundant. Shreds of false membrane may be present in the effusion.

Prognosis.—The fluid re-accumulates rapidly after puncture. It is rarely discharged through the bronchi, and is not often re-absorbed. The illness is usually of prolonged duration, from four to five months on the average. Relapses are of frequent occurrence, and the mortality is high (25 per cent., Straus).

Treatment.—Aspiration is useless. The ordinary operation for empyema should be performed.

Purulent tubercular pleurisies.

Etiology.—The attack may be preceded or not by pulmonary tuberculosis; but in the latter case there is generally tubercular disease of the bronchial glands. A purulent pleurisy in a tubercular subject is not necessarily tubercular, as it may be either of streptococcal or pneumococcal origin.

Morbid Anatomy.—The effusion is generally at first sero-fibrinous, and subsequently becomes purulent. The lesions may be older and more marked on the parietal than on the visceral layer, showing that the infection is not necessarily from the lung (Grancher). The pleura is often much thickened, and forms a fibrous shell encysting the exudation. The internal surface may be smooth or excavated by deep ulcers. The lung may be completely collapsed and lying by the side of the spine. There may be no tubercular lesions of the lungs: but if, as is generally the case, some are found after death, they are usually less advanced and of a less active type than in the opposite lung. Purulent tubercular pleurisies rarely discharge through the bronchi or by perforation of the thoracic walls.

Symptoms and course.—The onset and course may be acute or subacute, and the latter may be chronic. The duration is often very prolonged, and the general health may be fairly maintained in spite of considerable effusion, provided there be no fever or other symptoms which usually accompany empyema.

Treatment.—If the ordinary operation for empyema is performed, the cavity rarely closes owing to the great rigidity of the chest walls, and to the complete collapse of the lung. After aspiration the fluid is always, but usually slowly, reproduced. Unless there is urgent necessity it is, as a rule, better not to interfere.

Putrid pleurisy.—Effusions of this nature are invariably purulent. Streptococci and staphylococci may be found, but the putrescence is chiefly due to the action of saprogenic organisms, some of which are similar to those found in the buccal cavity (leptothrix, &c.) (Netter). Gangrene of the lung is often the cause of this condition, or the pleura may be infected by septic accumulations originating in neighbouring viscera.

Staphylococcus pleurisy.—The characters of the disease when originating from an infection by the *staphylococcus pyogenes* are given by Fernet, but owing to the frequency with which this organism is associated with others, and especially with the bacillus of tubercle, it is, as already stated, very doubtful, at least in purulent pleurisies, whether it should be regarded as the causative agent.

J. K. F.

CHAPTER LIII

SURGICAL TREATMENT OF DISEASES OF THE PLEURA

PAGE
Exploration of the chest 593
Paracentesis thoracis 595
Surgical treatment of empyema 597
    Steps of the operation 600
    Drainage tubes 602
    Washing out the pleura 604
    After treatment 605
    Method of closure of an empyema 606
(Subject continued in Chapter LIV.)

Exploration of the chest.—When physical examination has determined the probability of the presence of fluid in the chest, it is often desirable to verify the observation and to determine the nature of the effusion. Probably the commonest procedure is to insert the needle of a hypodermic syringe through an intercostal space. If this be absolutely the only instrument at hand it may be right to employ it, but it is very far from being well suited for the purpose. The needle is usually too short, for it must be remembered that the healthy chest walls are often as much as 1½ inch thick and not infrequently thicker, and if inflammation have occurred in the pleura they may be much thicker still. Again, this fine needle is easily blocked, and the suction power of the syringe is very slight.

It is far better to employ some form of aspirator, and by preference the bottle aspirator, and to attach to it not a simple needle but a cannula and trocar so arranged that the trocar can be replaced by a blunt plunger in order to clear it. The cannula should have a small lateral opening close to the end, so that in case a portion of lymph adherent to the costal pleura should have been detached and tend to fall back upon and close the terminal opening, the fluid may still flow through this lateral hole. The trocar should have only a short pointed end, not the long and useless point so much in favour with instrument makers. Sometimes it is useful to have a plunger of much smaller size than the cannula ; if this be inserted it will sometimes push away from the end such a mass of lymph as has been referred to, and thus the fluid can escape through the tube while the plunger is retained *in situ*. The cannula should not be too small; a diameter of $\frac{1}{10}$ inch is very suitable for the purpose, and it should not be less than 3 inches long for the exploration of the pleura, and decidedly longer if it be proposed to seek for fluid in the lung. It is a great mistake to suppose that the amount of pain caused is materially increased by using a cannula of this size; and provided that the point be really sharp and the end of the cannula fit properly on to the trocar, and the instrument be introduced quickly through an intercostal space without striking a rib, the amount of pain caused is really trifling, and it is not necessary to resort to the expedient of freezing the part or injecting cocaine. The first of these plans may render the accurate introduction of the instrument difficult, and the second may occasionally be followed by unpleasant or even serious toxic effects.

The air should only have been partially exhausted from the bottle; just enough indeed to produce a distinct negative pressure there. If a nearly complete vacuum has been made, the sudden rush of fluid into the tube is very likely to draw flakes of lymph into it and so obstruct the exit of the fluid. If indeed it be practically certain that the chest contains fluid, more information may sometimes be gained by the use of the exploring trocar than by that of the aspirator, but in doubtful cases the latter instrument should always be employed in order to render the introduction of air into the pleura impossible. A small artificially produced pneumothorax may do no harm to the patient, but it may seriously confuse the physical examination of the chest for some days afterwards.

If possible the patient should be in the recumbent posture, especially if it be proposed to extract any large amount of fluid, supposing such should be found.

No definite rules can be laid down as to the best position for the exploration; this must depend upon the peculiarities of each individual case. But it will appear from the sequel that if the chest is likely to be found full of serum, no better place can be chosen than the 6th or 7th interspace in the mid-axillary line; but if it be probable that the case is one of complete empyema the most convenient position for puncture is the 9th interspace, just outside the line of the angle of the scapula.

If the lung is to be punctured, the position of the heart and large vessels must be carefully borne in mind; and it must not be forgotten that dulness may be caused by the shrinking upwards of the lung, and that under these circumstances, if the lower part of the chest be operated on, the cannula may possibly be made to enter the liver on the right side, or the spleen, the stomach, or the splenic flexure of the colon on the left. In the long phthinoid chest the liver dulness reaches abnormally far above the lower margin of the thorax, and as this dulness varies somewhat with change of posture it may easily be mistaken for that caused by the presence of fluid in the pleura.

The precaution of purifying the skin of the part with 1 to 20 carbolic acid lotion should never be neglected, and the instrument used should always be carefully sterilised; but there is no need to leave any carbolic acid lotion in the tube of the aspirator. Such a procedure modifies the appearance of the first fluid drawn off, making either serum or blood assume the appearance of pus or pus mixed with blood. It is not a good plan to oil the instrument.

When the operation is concluded some antiseptic wool should be fastened over the puncture with flexible collodion.

Paracentesis thoracis.—If the chest be full of serum, which it is proposed to evacuate wholly or in part, the patient should be placed on the back very near to the edge of the bed, and the bottle of the aspirator having been partially exhausted and the skin purified, the cannula and trocar (which should not have a smaller diameter than $\frac{1}{10}$ inch) should be introduced, as described in the last section, in the sixth or seventh intercostal space in the mid-axillary line. This is the best situation because (1) it is the most convenient when the patient is in the recumbent posture; (2) the ribs are here least covered by muscle; (3) it is possible with a needle of suitable length to extract quite as much fluid as may be desired.

When the fluid begins to escape it should be allowed to flow until the stream slackens; then the exhausting syringe should be worked very slowly; just sufficiently fast in fact to keep the stream running. For it must be remembered that before long the dragging upon the lung will cause a fit of coughing, and the more rapid the process of exhaustion is, the more likely is this coughing to occur prematurely. In this way the rate of exhaustion can be regulated to a nicety, even more accurately than by adopting the syphon arrangement; a plan much vaunted by some, which will be described later. The amount of fluid to be withdrawn depends upon the nature of the case; but there are two occurrences that render the stoppage of the process imperative: (1) the onset of persistent pain and coughing, and (2) the appearance of blood in the fluid withdrawn. The coughing may sometimes be arrested by stopping the process of exhaustion for a time, or by giving the patient some ice to suck, after which it may be cautiously continued. In the meantime the pulse of the patient should be watched, and the position of the heart's apex noted. If the pulse show signs of failure it may be necessary to give some brandy. If the flow should be obstructed by the contact of the cannula with the surface of the lung or some flake of lymph, more fluid may often be obtained by passing the end of it back into the posterior and lower part of the chest cavity. When sufficient has been withdrawn some antiseptic wool should be fixed with collodion over the seat of puncture, and, if it is thought desirable, the chest may be strapped or bandaged, though it is very doubtful if this is of any use. An accurate note is then taken of the extent of the dulness, the position of the apex beat, and, in the case of the right side, of the position of the lower border of the liver.

Sometimes one hears of a 'dry tapping' or of the fluid ceasing to flow unaccountably, and this is occasionally spoken of as if fluid could possibly disobey the known laws of physics. This, it need hardly be said, cannot occur. There comes a time, indeed, when no more fluid can be drawn from the chest, although it is not empty except under the somewhat unlikely circumstance of the lung and the chest wall being so absolutely healthy that they can at once resume their normal relations. But as long as there is plenty of fluid in the cavity it must come out unless the cannula is blocked, or its point is raised above the level of the fluid, or unless the suction is insufficient to overcome the counteracting elasticity of the walls of the cavity in which the pleura lies. The suggestions made above with regard to the clearing of the cannula and lowering its extremity will, if followed, usually enable the operator to overcome these difficulties. But it must be remembered that the pleura may be in great part occupied by large masses of lymph which will give rise to all the physical signs which characterise the presence of fluid. These masses or old adhesions may divide the pleural cavity into compartments, so that it may be necessary to puncture the chest in more than one situation. It may indeed happen that the fluid extracted from one loculus may have quite a different appearance from that which is found in another.

Recently the suggestion has been made that inveterate cases of simple effusion should be treated by free incision, and some successful cases have been published. I think the cases must be very rare in which such a line of treatment is to be thought of: for the majority recover without it, and there must be a not inconsiderable risk of leaving a permanent fistula. Some of the most intractable ones are tubercular, and the surgeon is often asked to open these, especially if the fluid has become slightly turbid. My experience is that, if this be done, the fatal conclusion is hastened. Such patients are often able to get along very well with a very considerable collection of fluid in the chest; but when a free opening is made they become much more short of breath, and as the discharge is copious for a long time it is very likely that septic organisms will be allowed to enter. This is usually the beginning of the end. I have in a few tubercular cases which were, so to speak, halfway between serous effusions and empyemata, given considerable relief by inserting into the chest two cannulæ; one was inserted in front, which was made to communicate by means of an india-rubber tube with a large bottle of boric acid lotion made with warm sterilised water, and the other, which was inserted far back, was put in communication with the syphon or bottle aspirator. The fluid was gradually exhausted, and its place was taken by the boric acid lotion. As soon as the latter flowed quite clear into the bottle the anterior cannula was removed, and as much as possible was exhausted from the chest. This appears sometimes to diminish very considerably the rapidity of the reaccumulation. Possibly other fluids such as tincture of iodine and water might prove more efficacious.

The surgical treatment of empyema.—This section must be dealt with in several divisions. It will be necessary, before operating, not only to consider the age of the patient, but to endeavour by careful physical examination to determine what sort of a cavity is likely to be met with. Whether, for example, the empyema is general or localised, whether the pleura is thick or thin, whether the lung is adherent in places, or whether much contraction has occurred, as happens in old empyemata and results in a very close approximation of the ribs, and a drawing up of the diaphragm and the subjacent viscera into the position normally occupied by those of the thorax.

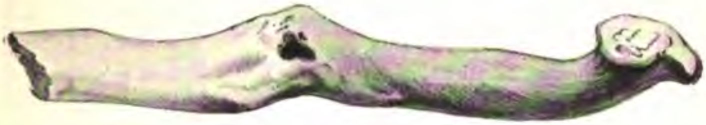


FIG. 133.—RIB SIX WEEKS AFTER A PART WAS REMOVED

Let us take to begin with the *uncomplicated general empyema of an adult.* The problem is to make an opening which shall not only be sufficiently free at the time of the operation, but which shall remain large enough until the time has come to remove the tube; and to make it in a position which will afford the best drainage both in the recumbent and in the erect posture.

Much unnecessary discussion has taken place on the question of the *removal of a portion of rib*, some writers speaking as if the operation presented serious difficulties, or were likely to be followed by inconvenience to the patient after healing has taken place. The removal of a short piece of a rib is not difficult, and it does not entail any material prolongation of the operation. It is not followed by any unpleasant consequences to the patient. A soft place is very occasionally left at the spot from which the rib was removed; but if this be the case the patient never complains of it. As a

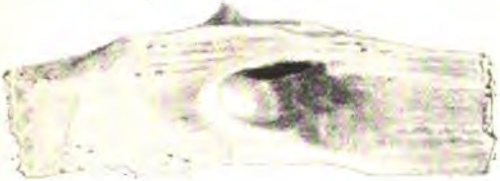


FIG. 134.—REGENERATED RIB AFTER A PART HAS BEEN REMOVED

rule the bone is regenerated only too fast. Fig. 133 shows the rib of a child who died of some other complaint six weeks after an operation for empyema. It will be seen that the rib, except for a little irregularity, is as good as ever. Fig. 134 and fig. 135 show the outer and inner aspects respectively of a rib, a portion of which had been removed, but the empyema had not closed, and well illustrate the bony track which forms round the drainage tube. A similar bony ring is formed if no bone has been removed, but in this case it is formed by two bars which are thrown out on each side of the tube and unite the rib above to the rib below. This is shown in fig. 136.

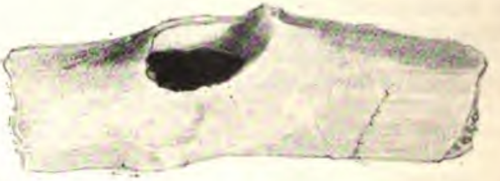


FIG. 135.—REGENERATED RIB AFTER A PART HAS BEEN REMOVED

Resection is recommended because it enables the surgeon thoroughly to explore the cavity with the finger and to evacuate the great masses of lymph so often met with, and also because it allows of the introduction of a large tube and diminishes the likelihood of nipping of the tube by the ribs as they draw together during the subsequent contraction of the chest. The bone is not removed with the idea of favouring this contraction: it can do nothing of the kind, and it is not essential nor indeed always advisable. The ribs may, for example, be so wide apart that the finger can be readily introduced between them, or the patient may be so very ill that even the short delay that is required for the removal of the portion of the rib is properly avoided. It may also be held that in very septic cases it is unwise to open the cancellous structure of the bone

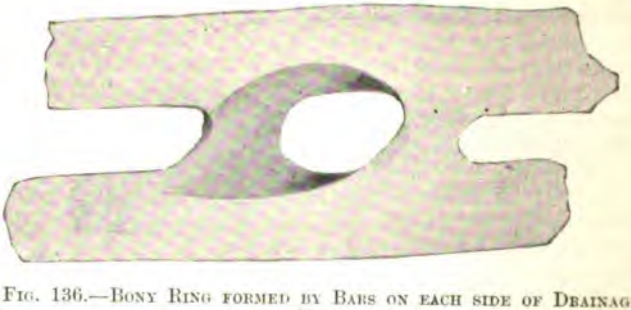


FIG. 136.—BONY RING FORMED BY BARS ON EACH SIDE OF DRAINAGE TUBE

to the discharges, though this argument is, I think, a weak one because under such circumstances the rib almost invariably becomes bared of its periosteum in a few days. I therefore confidently recommend the removal of a portion of rib as a routine practice to which there are certain exceptions.

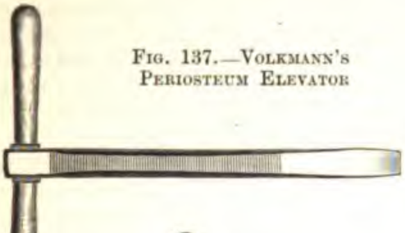


FIG. 137.—VOLKMANN'S PERIOSTEUM ELEVATOR

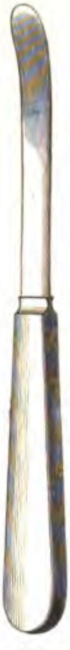


FIG. 138.—BLUNT PERIOSTEUM ELEVATOR

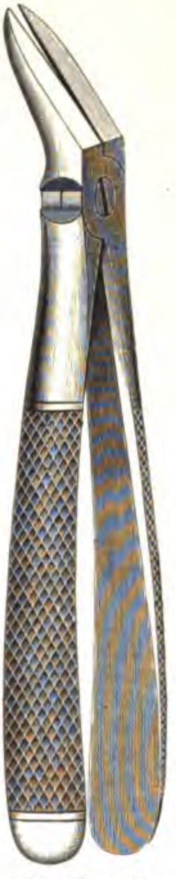


FIG. 139.—BONE FORCEPS SUITABLE FOR CUTTING RIB

The following *special instruments* will be found useful: a rather sharp periosteum elevator for separating the periosteum from the outer surface of the rib; that shown in fig. 137, invented by Volkmann, is a very convenient form: a blunt rounded periosteum elevator, slightly curved on the flat, fig. 138, for separating the periosteum from the deep surface of the rib. A large bent pair of bone forceps, fig. 139,[1] a large double blunt hook, and the other instruments which are required for any surgical operation, must, of course, be at hand.

Steps of the operation.—The skin of the part is purified, and the patient is anæsthetised, preferably with chloroform, and not very deeply. The patient is then brought very close to the edge of the table so that his side may project slightly beyond it, and is turned over very slightly on to his sound side—the less the better if the amount of fluid is great, so that the sound lung and the heart may be as little hampered as possible. The surgeon either stoops or seats himself on a stool by the affected side of the patient (fig. 140). The arm must not be raised above a right angle with the body lest the skin be drawn up and a valvular opening in the soft parts result. A mark should have previously been made on the ninth rib just outside the line of the angle of the scapula, and the presence of pus in this situation is now ascertained by the introduction of an exploring trocar. On no account should the chest be opened unless this is done. Before now the healthy side has been opened by neglecting this simple precaution.

The operator now steadies the skin with his left hand, taking care not to displace it either upwards or downwards, and makes an incision two inches or more long, with one or two strokes of the knife, straight down on the ninth rib. In this incision the periosteum must be divided. All bleeding vessels are now ligatured. The periosteum is then raised from the outer surface of the rib with the sharper of the two periosteal elevators, and from the deep surface of the rib with the blunter of the two instruments. It is important that this latter step should be carried out completely, as otherwise it is very likely that the pleura may be opened unintentionally and the later steps of the operation confused by the escape of pus. The large blunt hook is now applied to one end of the wound, and the soft parts are drawn forcibly in the direction of the long axis of the rib. The periosteal elevator is retained in position so as to render it easy to insert the blade of the bone forceps between the rib and the periosteum. The bone forceps are then pushed firmly towards the blunt hook, and the rib is divided. The same procedure is then carried out at the opposite end of the incision, an inch and a half or two inches of the rib being thus removed. Any further bleeding is now arrested, and then the pleura is opened by making a small incision with the knife above the level of the intercostal vessels, which is afterwards enlarged either with dressing forceps or with the finger. If the matter be septic, it may be thought a wise precaution to divide the artery and ligature it in two places in order to diminish the chance of secondary hæmorrhage. If this be done an attempt should be made to save the nerve, as otherwise the patient may complain afterwards of the patch of anæsthesia which results from its division. Before the pleura is opened, it is well to place over the side a large piece of lint or muslin which has been soaked in carbolic acid lotion (one to twenty). With the help of this it is easy to regulate the rate at which the pus escapes, a matter of some little importance, as collapse is sometimes the result of too sudden an evacuation of a large quantity of fluid. This precaution has the incidental advantage of keeping the escaping matter within reasonable bounds.

---

[1] Many special forms of bone forceps and rib scissors have been invented, but the ordinary pattern will answer every purpose. I have devised one myself but have discarded it in favour of the instrument which every surgeon is sure to possess.

If the chest be very full of fluid, and the breathing and the heart's action very much hampered in consequence, it is often advisable to remove a considerable quantity of the pus by means of the aspirator before administering the anæsthetic; and, if the removal of a piece of rib has been decided upon, to postpone this part of the operation until the matter has been allowed to escape through a free opening in the intercostal space.

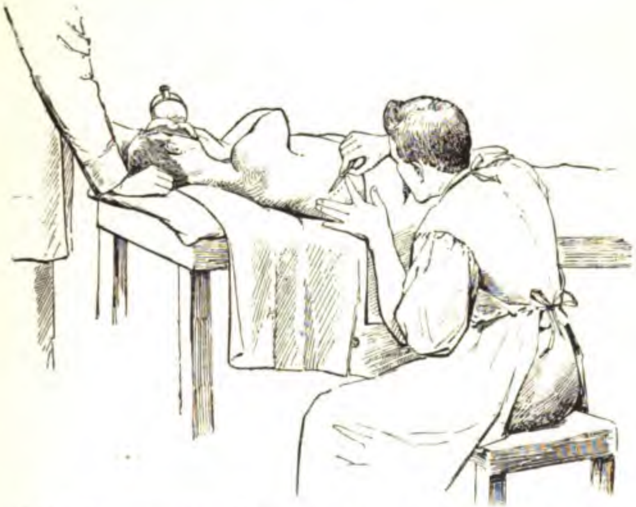


FIG. 140.—OPERATION FOR EMPYEMA: THE PATIENT LYING ON THE BACK

Another method of proceeding is to place the patient on the affected side, and to operate from behind as shown in fig. 141.

Before the pus is allowed to escape, the finger should be inserted so as to ascertain the degree to which the lung is collapsed, and the amount of coagulated lymph which is present. At the conclusion of the process the finger should be inserted again, so that an opinion may be formed as to the amount of expansion of which the lung is capable. At this second exploration it will be more easy to ascertain how far the lung is adherent to the diaphragm and the chest wall, an observation of some importance, as on the presence of these adhesions depends to some extent the chance of a speedy recovery. All the coagulated lymph having been extracted by the finger, and as much as possible of the pus, by turning the patient over on to the affected side, the drainage tube is inserted and the dressing is applied. No stitches are to be used.

Countless forms of **drainage tube** have been devised at different times, of which it may be said that the simplest is the best, and it is interesting to note that a drawing of one almost precisely similar to that I recommend is to be found in the works of Ambroise Paré. This particular form was devised by the late Dr. Buchanan Baxter of King's College Hospital. Anyone can make them for himself out of a piece of ordinary drainage tube, india-rubber sheeting of one-twelfth of an inch in thickness and a piece of silver wire. Their construction will be sufficiently explained by the accompanying figure (fig. 142) without any detailed description. The size should not be smaller than that of the little finger (diameter ½ inch). The tube should be sufficiently long to project into the chest for about an inch or less, and should have one hole lateral only, close to the extremity. It is worse than useless to have a long tube coiled up in the chest, but if it do not project a little into the pleural cavity, it is apt to be pushed out by the movements of the chest walls. There is no dressing, I think, better than the double cyanide gauze, which should be used in considerable quantity, and outside this a mass of some antiseptic wool, secured with a bandage. The use of an extremely soluble antiseptic, such as sal alembroth, is to be deprecated, as where the discharge is likely to be very copious, for obvious reasons irritation is apt to occur at the margin of the dressing. In stinking cases it is needless to use such expensive dressings. A few thicknesses of boric lint soaked in sublimate lotion (1 in 2,000), and outside this a mass of oakum or wood wool, forms a very admirable application.

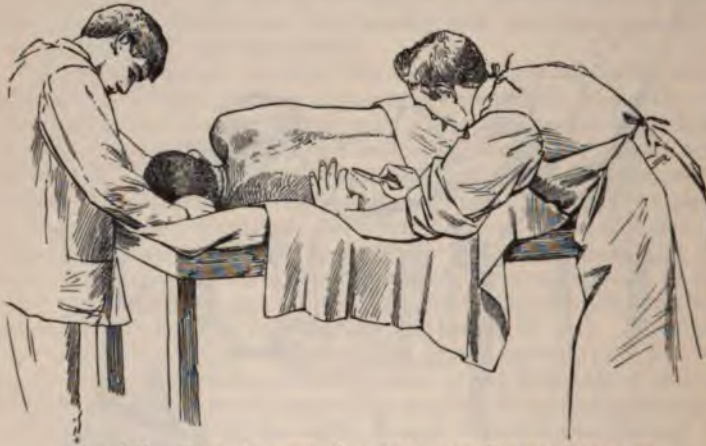


FIG. 141.—OPERATION FOR EMPYEMA: THE PATIENT LYING ON THE AFFECTED SIDE

Besides the bleeding from the wound, there is sometimes considerable *hæmorrhage* from the inside of the pleura. This is precisely analogous to that which is met with in opening large abscesses, such as some of those connected with the spine. It may be very severe, and indeed dangerous. In a spinal abscess I have seen it prove fatal. Unfortunately, nothing can be done for the arrest of this bleeding, unless the cavity be small enough to admit of efficient plugging, but in most cases it stops without any serious consequences. Should the bleeding from the wound be difficult to arrest, and especially if the patient be faint at the time, it will be advisable to plug it firmly round the tube. Plugging round the tube is also wise when the matter is septic. In this case some enduring antiseptic, like chloride of zinc solution (40 grains to the ounce), or iodoform, or both, should be used on the plugs.

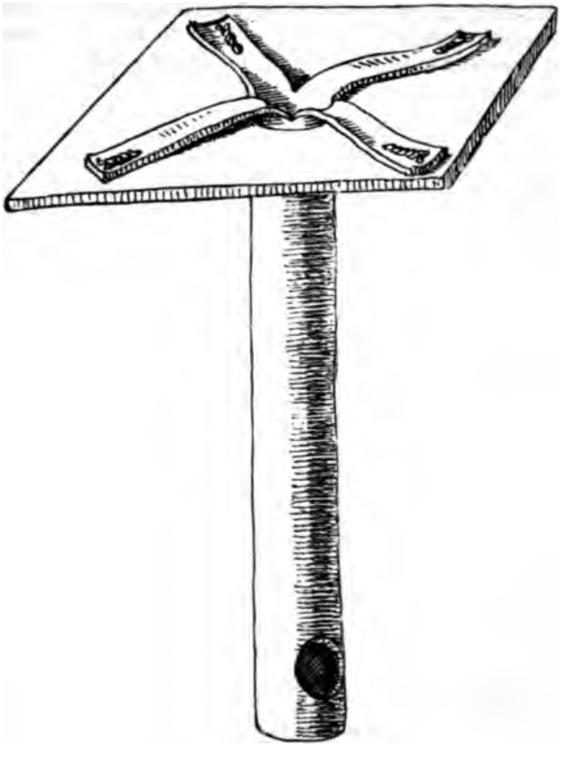


FIG. 142

It may be asked why the ninth rib just outside the line of the angle of the scapula is chosen as the seat for the incision. Others have been recommended at different times. The old seat of election was the sixth or seventh interspace in the axilla, a spot which was selected because of the comparative freedom from muscular covering. Mr. Marshall used to recommend the fifth interspace close to the edge of the pectoralis major as an appropriate place, because he said that spontaneous rupture of empyemata most commonly occurred at this spot. This, which, by the way, is open to some doubt, is no proof that the point of spontaneous rupture is one that will afford the best possible drainage. Good enough drainage may no doubt be obtained from the axillary incision when the patient is standing upright, but, as long as he maintains the recumbent posture, a certain amount of fluid is sure to collect in the hollow formed by the angle of the ribs. The spot I recommend allows perfect drainage when the patient is recumbent, and also when he is standing upright. It may also be asked how this can be, seeing that the normal pleura extends very considerably below the level of the ninth rib. But it is almost invariably the case that, as the lung shrinks away, the diaphragm becomes adherent to the inside of the chest walls for some considerable distance, and the lower part of the pleura is, in consequence, obliterated. Even if this have not occurred before the pus is let out, it almost always happens before long, as the result of the efforts of nature to close the cavity. If, therefore, the extreme lower limit of the pleura be selected for the incision, it will be found after a time that the track leading into the main cavity becomes longer and longer, and more and more oblique, so that the difficulty of retaining the drainage-tube is increased, and the necessity of making a higher opening not infrequently arises. I have often been called upon to do this, and sometimes to make a posterior incision in cases where the pleura has been opened either spontaneously or by the surgeon in front or at the side, but I have never had to incise the pleura in front in cases where the posterior incision had been made to begin with.

This is an appropriate place to discuss the question of **washing out the pleura**. Some surgeons do it as a matter of routine when the pleura is first opened, and others carry it out at stated intervals in the subsequent treatment of the case. My opinion is that it is seldom necessary, and, if not necessary, that it is better to avoid a procedure which is not unaccompanied by risk, however remote. The most stinking empyemata, if simply opened, usually become sweet in the course of two or three days, and I have never seen anything in published accounts to suggest that other not obviously septic cases behave better as the result of irrigation. The danger associated with washing out is that of sudden death from causes not at present ascertained. That this danger is a real one may be gathered by reference to many published cases (*vide* 'Trans. of Clin. Soc.' vol. x., by Dr. Cayley), and the cause of death not being known renders it impossible to guard against this with certainty. I once was present at one of these fatalities. It occurred, in my student days, to a little boy with a small left localised empyema with dense fibrous walls. It was the custom to wash him out daily with diluted tincture of iodine as he sat up in the hospital ward, and it was during one of the daily washings that he suddenly expired.

Sometimes however, as, for example, when the discharge keeps offensive or remains purulent for a very long time, it is advisable to resort to flushing out of the cavity. The following precautions should then be adopted. The patient must be kept in the recumbent posture, the large tube should be retained in the wound, and through it should be inserted a tube of much smaller size (Jaques' catheter, about No. 8, is very useful for the purpose), which is attached by means of a long tube to an irrigator. The irrigator should not be raised more than eighteen inches above the level of the patient's chest, and a careful watch should be maintained in order to see that the fluid escapes as fast as it is allowed to flow in. If any doubt is felt about this, the small tube should be withdrawn, and the patient should be requested to empty the chest by coughing. I have never seen any evil consequences happen when this plan was adopted.

After treatment.—When the dressing has been applied the patient is put to bed, and it must not be forgotten that considerable shock sometimes follows the operation. In some cases, especially in small children, this has been known to prove fatal, and not infrequently it is necessary to have recourse to pretty free stimulation. The tendency to collapse probably depends partly on the loss of blood and partly on the altered respiratory conditions, often very irksome at first, and to which the patient has gradually to become accustomed.

It will very likely be necessary to change the dressing for the first time at the end of a few hours. Afterwards once a day while the discharge is copious, and less frequently as it diminishes, will be sufficiently often. In changing the dressings it is well to replace the first piece of gauze, soaked in (1 to 20) carbolic acid lotion, as quickly as possible, so as to prevent the unnecessary entrance of air (with whatever dust it may contain) into the pleura during the movements of respiration. In septic cases the dressings must be changed much more frequently—three or four times in the day, or oftener—until the discharge becomes sweet. The tube may be left *in situ* for a week, at the end of which time it will have formed a smooth track for itself, and can be removed and reintroduced without trouble to the patient. If, as sometimes happens, the patient finds it difficult to move, a many-tailed bandage may be substituted for the ordinary roller, but the latter keeps the dressings more securely fixed.

The patient should be kept in bed in all cases for about a week, and occasionally for a much longer time. But if at the end of a week the temperature is normal, and he is doing well, the assumption of the erect posture for some part of the day, and the more free movement of the chest which results, can only be beneficial. It is not necessary to keep indoors all the time that the tube is being worn.

No hard-and-fast rule could be laid down as to the length of time the tube is to be retained; each case must be judged by itself, the length of time depending principally upon the amount of discharge. In small children a few days is generally sufficient, and many cases have been known to do well when the tube is removed on the second day. In adults a longer period is usually required, but even in those that are progressing most favourably it is seldom wise to remove it until three or four weeks have elapsed. It is not always necessary to wait till the cavity is reduced to a small size, provided that the discharge be slight and of a serous character.

I have often succeeded in finishing off a case of this kind, which would otherwise have been tedious, by adopting the following method of treatment. The tube is removed, and the external opening is allowed to close with granulations. At the end of a certain period, depending upon the amount of previous discharge, say two or three days, a railroad catheter is pushed along the sinus, and the fluid that has accumulated is drawn off. If this be serous and not more than a few ounces, another interval of a few days is allowed to elapse, and the same process is repeated. If at the end of this time the accumulated fluid is still serous and less in quantity, a still longer interval may be allowed to elapse, and when after several days it is reduced to a few drachms, the pleura may be safely trusted to deal with it. Should, however, the accumulation remain or become purulent, resort must be had again to the drainage tube.

Method of closure of an empyema.—This leads to the consideration of the manner in which, under ordinary circumstances, closure of an empyema takes place. No inflation of the affected lung can occur during the ordinary process of respiration. It is only when the mouth is closed, and an expiratory effort is made, that a certain amount of air can be pressed into it from the opposite side. The cavity must therefore be closed:

(1) By such expansion of the lung as may depend upon its natural elasticity, which has been previously overcome by the accumulation of fluid, and which probably is greatest when the lung has been consolidated by pneumonia.

(2) By the falling in of the chest walls.

(3) By the drawing upwards of the diaphragm, outwards of the mediastinum, and downwards of the structures of the posterior triangle.

(4) By the contraction of the granulation tissue lining the pleura, which not only affects this drawing inwards of the various structures bounding the pleural cavity, but also is constantly drawing the lung out to meet them. It is because there often comes a time when as much contraction of the surrounding parts and expansion of the lungs as possible has taken place, that the process of closure comes to a standstill.

(5) When the method described above of periodical tapping with the railroad catheter is adopted, another force is introduced to aid in the closure of the cavity. This is the absorption of air

by the pleura. That such absorption takes place in cases of traumatic pneumothorax is well known, and that it may occur in cases of empyema is proved by the fact that not infrequently at these periodical tappings a negative pressure is found to exist.

It has been hinted that in a certain number of cases of empyema the cavity does not close as the result of a simple incision; this depends upon the changes that take place in the pleura itself, in the lung, and in surrounding parts, the discussion of which conditions may be conveniently considered here.

In an *empyema of old standing*, whether it has been opened or not, the pleura becomes enormously thickened; it becomes converted indeed into a thick layer of fibrous tissue, which it is not uncommon to find half an inch in thickness, and often much more than this. The fibrous tissue cuts with a gristly section and is intensely hard; and, as in obedience to the universal law mature fibrous tissue occupies a smaller space than the granulation tissue from which it is formed, a large amount of contraction must necessarily occur during the process of its development. This contraction reacts in various ways upon the surrounding parts. Thus the lung tends to become more and more compressed, and its functions are more and more interfered with in consequence. The parts that surround the pleura are being constantly drawn towards one another, so that the arch of the diaphragm comes to occupy a higher position than normal, and there is a corresponding displacement upwards of the higher abdominal viscera, whilst the mediastinum is drawn more and more to the affected side. The changes that take place in the parietes of the thorax over and above those that depend on their increased thickness result in the approximation of the ribs to one another, and in the development of lateral curvature of the spine. This lateral curvature, like the ordinary scoliosis, is accompanied by torsion of the vertebræ, but it differs from it in certain obvious characteristics. In the first place, the curve is, as a rule, much longer, and extends from the lower part of the cervical region to the lumbar region (fig. 143). The compensatory curves are proportionately shorter. In the next place, the shape of the chest differs from that met with in scoliosis, inasmuch as on the contracted side it is flattened both in front and behind, whereas in scoliosis flattening is met with in front, on the same side as that on which the angles of the ribs and the angle of the scapula form the characteristic gibbosity or hump. The ribs, again, do not take the same shape as in scoliosis, but are simply drawn very closely together; not uncommonly, indeed, they overlap one another, and sometimes several ribs have been found united by cross-bars, or into one solid mass. The patient is often alarmed at observing drooping of the shoulder which is caused by this lateral curvature, though to the surgeon it is an augury of success. He is often asked whether it is to be permanent; to this he should give a guarded reply. But it may be safely said that in children, if the curvature be not great and if the cavity close within a reasonable time, almost complete recovery is likely to occur. Not so, however, where the closure is long delayed, or in adults. He may, however, console his patient by telling him that a certain amount of drooping of the shoulder is very inconspicuous, and that a very large proportion of healthy people really possess the same deformity to a small degree.

Another change that is met with in the pleura results from the deposit of *calcareous plates*. These are sometimes small and sometimes of a very large size, and, as far as I have seen, are most frequent in tubercular cases. The tendency to the formation of these calcareous plates is most inveterate, and if they are met with on opening the empyema the chance of complete closure is very small. If the plates are large and fixed it is not wise to attempt their removal, as it may be impossible to tell how far they extend, and what large and important structures may be injured by dragging upon them.

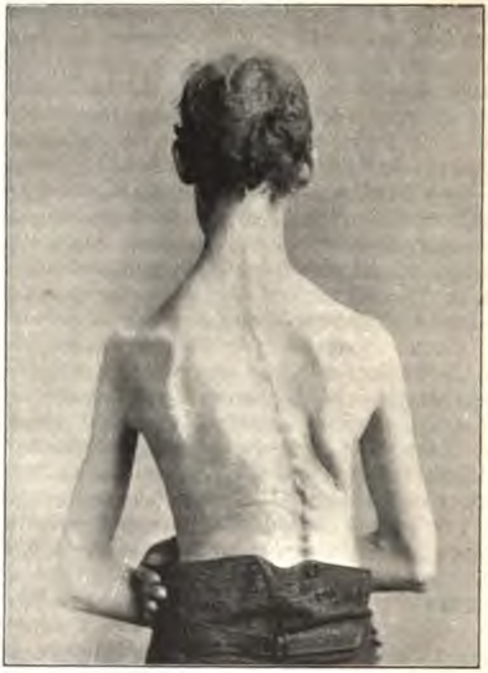


FIG. 143.—LATERAL CURVATURE FOLLOWING EMPYEMA

If an empyema does not close either on account of imperfect opening or in spite of the freest drainage that can be obtained by a single or double opening, certain other constitutional changes are likely to occur, but they are more frequently met with where the exit for the discharge has been imperfect than where it has been free. They do not differ from those which are produced by chronic suppuration elsewhere. The patient becomes pale and anæmic, has a more or less hectic temperature, and in time develops amyloid changes in the viscera which are recognised by the presence of enlargement of the liver, pale and perhaps albuminous urine of low specific gravity, diarrhœa, emaciation and clubbing of the fingers and possibly of the wrists and nose. It is in cases of empyema that the most striking examples of the rapid disappearance of those conditions are noted as soon as the suppurating cavity has been made to close. I have seen a young man with an imperfectly drained empyema which did not contain more than a few ounces of pus rapidly develop a liver which extended below his umbilicus, whilst his fingers were clubbed and his urine was solid with albumen on boiling. A few weeks of satisfactory drainage caused the closure of the cavity, and in considerably less than two years the liver dulness was normal, the clubbing had disappeared, and there was only an occasional trace of albumen in the urine.

R. J. G.

CHAPTER LIV

SURGICAL TREATMENT OF DISEASES OF THE PLEURA

(continued)

PAGE
Estlander's operation 610
Varieties of empyemata 613
Localised empyemata 613
Double ,, ... 614
Prognosis in empyema 614
Spontaneous rupture 615
Rupture into a bronchus 615
Mode of death in empyema 616
Cerebral abscess 616
Difficulties in diagnosis 617
Sub-diaphragmatic abscess 617
Suppurating hydatid 618
Pyopericardium 619

Estlander's operation.—In the year 1877 an operation was devised by Estlander, of Helsingfors, for the relief of cases in which simple drainage proved ineffectual. Recognising that non-closure depended upon the fact that contraction of the surrounding parts and expansion of the lungs had reached their utmost possible limits, he endeavoured to enable further contraction to occur by removing considerable portions of the unyielding structures of the chest wall. His plan was to take away as much of the ribs as corresponded to the outer wall of the cavity.

The steps of a so-called Estlander's operation will now be described. The surgeon in the first place endeavours to ascertain by means of a long probe (pewter probes are very useful for the purpose) approximately the size of the cavity. He plans his incision in such a way that free access is gained to all the ribs corresponding to the cavity, whilst inflicting as little injury as possible upon the muscles and avoiding the section of important nerves. Thus, for an example, an S-shaped incision, or a Z-shaped incision, or a round or a square flap will often enable him to turn forwards the pectoralis major, or backwards the latissimus dorsi, or outwards the scapula, whilst avoiding the long thoracic or the spinal accessory nerve. If it be only required to remove three ribs, a single incision in the direction of the central one will generally be found sufficient for the purpose. The incision is carried down to the bones, and the soft parts are rapidly reflected from them. The periosteum being left *in situ*, a portion of rib or ribs in the neighbourhood of the sinus is then removed, sufficiently large to enable the finger to be introduced, for which purpose it will also be necessary to make an incision in the thickened pleura. Some difficulty is likely to be met with in this stage from the fact that new bone has been most probably thrown out, uniting the ribs together, and forming a bony ring round the sinus which has been occupied by the drainage tube (figs. 134, 135, 136). After the introduction of the finger a more accurate idea is obtainable of the extent of the operation which will be required. This having been ascertained, each rib is removed in the same way as described on p. 600. For, although it is not proposed to preserve the periosteum, it is a simpler and quicker process to separate it from the ribs than to take it away with them. The next step is to remove the thickened pleura and the intercostal muscles. This may be done freely with a pair of strong curved scissors; and it is interesting to observe that at this stage the hæmorrhage is slight, although in the preliminary process of clearing the ribs it is often troublesomely free. This appears to depend upon the fact that the intercostal arteries have been practically obliterated by the pressure of inflammatory products, their functions having been taken on by smaller and more superficial vessels. It may even happen that no intercostal vessel at all has to be ligatured, but the surgeon must be prepared to secure any vessels that bleed. There is now left a flat surface at the bottom of the wound, which may be either dressed from the bottom by plugging it with antiseptic gauze, or, if it seems advisable, the skin may be partially stitched up whilst a drainage tube is inserted at a convenient spot. As a rule, it will be found that the upper part is the most difficult to close, and it is here, therefore, that the drainage tube will probably have to be placed. It is this no doubt that accounts for the fact that Estlander's operation, whilst sometimes successful, often leads to great improvement, but leaves a sinus at the upper part of the chest. It is open to question whether it is justifiable to remove part of the second rib, and it is extremely doubtful whether the first rib should ever be submitted to such a process. No doubt most of the failures depend upon the insufficient removal of ribs. When all has been said that can be said against this operation, however, it must be recognised as a real advance in surgery. Fig. 144 is taken from a patient on whom a considerable operation of this sort had been done, but in whom the cure was not complete. A back view of the same patient is shown in fig. 143.

It has been suggested that, before applying the dressing, the remaining part of the pleura should be scraped; but it is not clear that this is really beneficial, and it must not be forgotten that if it be done completely, the sharp spoon may be applied very close to the important structures occupying the mediastinum. An attempt may, however, be made to render the cavity aseptic by the application to the surface of a solution of chloride of zinc (40 grs. to the ounce) and the employment of a limited quantity of iodoform.

The operation is not lightly to be undertaken, as it involves a considerable amount of shock and loss of blood, and patients have been known to succumb to it. I have heard of a patient dying during the process of irrigating the surface of the pleura before stitching up the wound. It must also be remembered that these cases are nearly always septic, and that a large area of cancellous bone is being exposed to the action of septic discharges. It is truly remarkable how slight is the deformity after very extensive operations of this sort—often much less than is met with in cases that close by natural processes. This is probably accounted for by the fact that it is precisely in those cases in which contraction does not occur that Estlander's operation is called for.

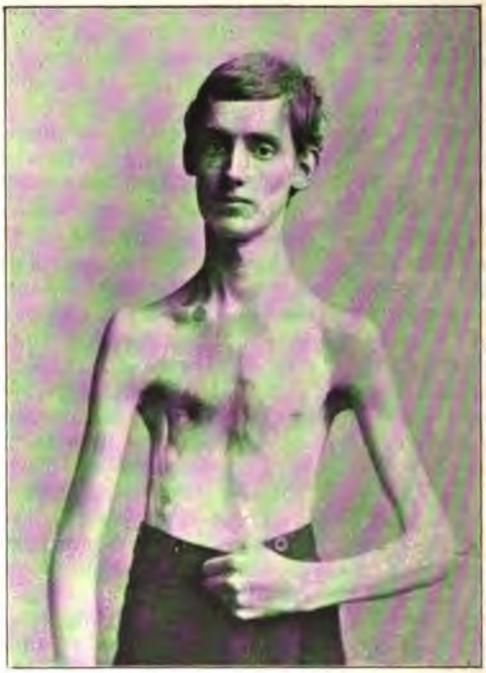


FIG. 144.—RESULT OF ESTLANDER'S OPERATION

Varieties of empyemata.—It is unusual to meet with a genuine *general empyema*. It seldom occurs except in those cases where the introduction of septic matter into a healthy pleura has given rise to the rapid effusion of purulent fluid into it. Such cases are those of rupture into the pleura of sub-diaphragmatic abscess, or of liver abscess, or of a pericæcal abscess, or of a septic abscess of the lung or abscesses which have been caused by external wounds; but perhaps the most common of all are those empyemata which have been caused by the introduction of an exploring needle through a healthy pleura into a septic lung. I have myself produced this result in exploring a case of bronchiectasis, and I have operated upon one which was caused by the exploration of the lung which had become gangrenous as a result of the pressure of an aneurysm on the left bronchus.

In the great majority of inflammatory cases, however, some parts of the lung are adherent to the parietal pleura. The most common situations for these adhesions are between the base of the lung and the diaphragm, between its inner surface and the pericardium, between the back of the lung and the hollow formed by the angles of the ribs, and at the apex. It will thus be seen that most empyemata are in fact circumscribed, but the term *circumscribed* or *localised empyema* is, as a rule, reserved for those cases in which the suppurating cavity occupies only a small part of the pleura.

Localised empyemata may be conveniently divided into three classes. The first, and by far the commonest, includes those in which the matter is contained in a cavity between the outer surface of the lung and the costal pleura. The second variety is that between the diaphragm and the base of the lung, the so-called *diaphragmatic empyema*, and the third is between two contiguous lobes or between the lung and the mediastinum. The treatment of the first offers no special difficulties beyond those of diagnosis. Both diagnosis and treatment are more difficult in the others, and, indeed, no rules can be given for the treatment for the third class of cases. If there be a reasonable prospect of finding matter between the diaphragm and the lung, this region may be safely explored after removal of a portion of rib, preferably the ninth, midway between the axillary folds, care being taken that the portion of rib removed is above the level of the lower limit of the pleura. The finger introduced through this opening may be readily made to pass between the lung and the diaphragm, and can explore almost the whole surface. But, if it be not long enough, the investigation may be completed with a blunt instrument slightly bent upon the flat.

There may be more than one localised empyema in the same patient. It may also happen that a large cavity is incompletely subdivided into loculi by the presence of adhesions. In such cases it is often difficult to obtain satisfactory drainage. The prospect of complete closure is greater in loculated than in general empyemata; but, if this result be not obtained, resort may be had to Estlander's operation with considerable confidence of success.

The question of the proper treatment to be applied to **double empyema** has been much debated, some surgeons preferring to operate upon both at the same time, whilst others consider that at least some days should be allowed to elapse between the two operations. It used to be considered impossible that the patient could breathe with both his pleural cavities open, and this would be the case if there were no adhesions. The presence of the adhesions permits the movements of the chest to cause a certain amount of expansion in the lungs, and this accounts for the fact that cases may do well in which both pleuræ are open at the same time. I cannot but think, however, that it is safer to allow the patient to become accustomed to the altered respiratory conditions which are involved in the opening of one pleura, and which, as has been said, often make respiration for a time very difficult, before interfering with the opposite lung. Of course there may be cases in which both sides are so full that it is thought best to avoid any delay; but even in these I think it would be safer to open one and aspirate the other, and to reserve the completion of the operation for a later occasion.

Prognosis.—It is impossible to make a comprehensive statement about *prognosis* in cases of empyema, seeing that they depend upon such a variety of causes, and that the chance of recovery is also dependent on so many conditions. In a general way, however, it may be said that in acute empyema of children and young people prognosis is very favourable, and that even in older people and in chronic cases, provided the lung be not inordinately compressed, there is very fair chance of recovery, even if the opening have been long delayed. The points that militate most against success are the absence of adhesions, rigidity of the chest and great collapse of the lung, the tubercular taint, and, under some circumstances, the formation of a bronchial fistula, or the presence of some permanent source of irritation such as a carious spine or rib, or that of a tumour or of a foreign body. Of all these the tubercular element is perhaps the most unsatisfactory. It usually leads to disappointment in what otherwise appear to be most promising cases. Should suppuration persist there is nothing for it but for the patient to go on wearing the tube; fortunately, however, it is possible to live a happy life under these circumstances. I am acquainted with a tubercular patient who has worn a tube for seventeen years. He is in active business, and during the last few years has married. His cavity is small, but will not close in spite of the performance of a very complete Estlander's operation. Another patient, one of the first I ever operated upon more than twenty years ago, making the comparatively small opening which was common in those days, has gone on wearing his tube ever since because he could not make up his mind to submit to another operation with a view to obtain better drainage. His fingers have become a little clubbed, and he is pale, and there is a little albumen in the urine; he has also suffered from an inter-current attack of syphilis; still he is, or was recently, alive, following the active occupation of a costermonger. Such facts as these are interesting to those who may have to answer questions with regard to prognosis. The patient with a permanent fistula should have two tubes which he should use on alternate days, keeping the one he is not using in 1 to 20 carbolic acid lotion. A few thicknesses of boracic lint and a pad of wood wool will be found a very satisfactory dressing. No effort should be spared to maintain, as far as possible, asepsis in these cases.

Spontaneous rupture.—A previous reference has been made to the spontaneous bursting of empyemata, and it was stated that the late Mr. Marshall maintained that the most usual place for spontaneous rupture was in the fifth intercostal space, just outside the edge of the pectoralis major. Cases of spontaneous rupture are not sufficiently common to enable many surgeons to form an opinion of their own on the question. I have seen it occur through an upper interspace close under the clavicle, through one of the middle inter-spaces near the sternum (which, it will be remembered, may be in front of the pericardium), occasionally behind the breast or in various situations in the back, even as far down as the tenth or eleventh interspaces. Still, I believe that, on the whole, Mr. Marshall was right in his assertion. This, however, by no means concludes the list of possible situations for external rupture; pus from the pleura may find its way like a psoas abscess into the abdomen, and may point at the groin, in the thigh, or, it is said, even at the knee or the ankle. I have not myself seen an empyema reach lower than the iliac fossa, and even in this case the diagnosis was not quite certain. Whether Nature's opening, in a case that has ruptured externally, should be trusted to carry on the drainage depends on its size and situation. Generally, however, it will be found that Nature is not a satisfactory surgeon, and that it is wisest without delay to make a second opening in the best situation. One of the most peculiar cases of spontaneous rupture that I have seen occurred in a middle-aged man after influenza--an abscess developed almost in front of the axillary vessels below the clavicle; it had been discharging for some time when he came under my care, and dulness was almost confined to the supra-spinous fossa. After making an incision in order to follow down the sinus I was obliged to draw the axillary vessels outwards, and open the empyema in the first intercostal space. This led to a rapid and complete recovery.

Rupture into a bronchus.--Far more common than external rupture is the rupture of an empyema into a bronchial tube. Its occurrence is readily recognised by the sudden onset of cough and expectoration frequently preceded by a slight hæmoptysis; this is often extremely distressing to the patient, and in very rare cases may prove fatal by causing suffocation. Opinions are divided as to what should be done under the circumstances; it is probably wisest to watch the patient carefully for a few days, and see whether the discharge diminishes, whilst, at the same time, the temperature keeps down. Should this, however, not be the case, it implies that the opening is not sufficiently free, and then nothing is to be gained, whereas much may be lost, by delay in making an external incision. The danger is not only to the affected lung, but also to that on the other side, from the inspiration into the bronchial tubes of pus from the pleura. It is unfortunately not uncommon for cases of this sort to be allowed to drift on from week to week, and from month to month, and even from year to year in the hope that in the course of time the cavity may close. In the meantime, not only does the lung suffer, but amyloid changes are imminent, and the chances of a cure, when operation is at last resorted to, are greatly diminished. It has been said that in some cases the presence of a bronchial fistula prevents the closure of an empyema; this appears to depend upon the fact that the fistula is very large, under which circumstances its existence can be recognised, in the first place, by the loud sound that is made when the patient draws his breath, and in the second place by the fact that, if the mouth and nose be closed, he can draw air into the opposite lung, as may be proved by listening with a stethoscope, and, in the third place, by the mucoid character of the discharge. I have under observation a patient who is in robust health, but who will apparently have to wear a tube for the rest of his life from this cause. It must not, however, be supposed that in such cases closure can never take place; it may occur, provided the cavity of the empyema has been abolished. If nothing except an opening direct into the bronchus is left, and should the discharge become infinitesimally small, it is often safe to take out the tube, when the external wound will probably close rapidly. But such a result can hardly be anticipated if the bronchi in the affected lung have become dilated and secrete more than the normal amount of mucus. I have heard suggestions for the removal of a part or the whole of such a diseased lung under these circumstances, but have not myself attempted such a heroic line of treatment. It seems indeed open to question whether so dangerous an operation is justifiable.

Mode of death.—An empyema may cause death in various ways. If unopened, the patient may die from interference with the respiration. Within a short time of the operation he may die of shock, or loss of blood, or subsequently from secondary hæmorrhage, from the exhaustion produced by long discharge from acute albuminuria, or from blood-poisoning. In the last category must be included those not very uncommon cases in which an empyema or a pulmonary abscess has been followed by abscess in the brain. Why this should be so, comparatively speaking, common is quite unexplained. Sir William Gowers,[1] quoting from Martin's 'Hirnabscesse' (Berlin, 1892), says that this form of cerebral abscess is called 'pulmonal cerebral abscess.' He adds that 'they never result from true tubercular cavities, that the abscess is single in about half the cases, and is generally situated in the cerebral hemispheres, especially in the posterior lobe. The cerebellum is not often affected from this cause, and never suffers alone.' R. Nather[1] says that cerebral abscess occurs in about eight per cent. of cases of such diseases as gangrene of lung, fœtid bronchitis, and bronchiectasis. His statistics are compiled from ninety-eight cases.

---

[1] *Diseases of the Nervous System*, vol. ii. p. 477, second edition, 1893.

These abscesses are seldom amenable to surgical treatment. I have opened one without good result, probably because it was not single, and I have searched for one which appeared to give positive evidence of being situated in the motor area, but was really in the occipital lobe. Whilst writing this chapter I was hesitating whether to explore a case without localising symptoms, in which after death no less than seven separate foci of suppuration were found. Still, if distinct localising symptoms are present, the surgeon's duty is clear: he must endeavour to open the abscess, for, if unrelieved, the fatal result is inevitable.

Difficulties in diagnosis.—Even after the discovery of pus by the introduction of the exploring needle through an intercostal space, it is often impossible to say whether it is in the pleura or elsewhere, and this difficulty is not always cleared up when a free opening has been made and the finger has been introduced. It may, for example, happen that the needle has entered an abscess or a suppurating hydatid in an adherent lung, or it may pass through the diaphragm into a sub-diaphragmatic abscess, or into an abscess or suppurating hydatid of the liver, or it may have been made to enter the pericardium or an abscess situated between the pleura and the rib. These various conditions must therefore now be shortly considered.

Abscess of the lung and hydatid of the lung are treated of in Chapter XIX., p. 241, and in Chapter XLI., p. 482, to which the reader is referred.

Sub-diaphragmatic abscess may arise from a variety of causes which may be considered under two heads: (1) *intraperitoneal*, namely, those originating from the stomach, the duodenum, the transverse colon, the liver and the spleen, &c.; and (2) *extraperitoneal*, which start from such structures as the kidneys, posterior part of the liver, cæcum, the vertebræ and the ribs. In cases of doubt, therefore, inquiry should be made as to the existence of previous disease in any of the parts mentioned. By far the most common causes of such sub-diaphragmatic abscesses as are likely to lead to confusion in diagnosis are the gradual rupture of an ulcer in the upper part of the stomach, or in the transverse colon, or the bursting of an abscess of the liver; they do not of course involve the whole peritoneal cavity, but are limited by adhesions at their lower parts. If the abscess have burst from the liver, in all probability that viscus will have been pushed very considerably downwards, but the limit of dulness upwards will have been increased in still greater proportion. If it have originated from a perforation of the transverse colon or the duodenum, it will probably contain gas, and a tympanitic area will then exist corresponding to the size of the cavity, abolishing the liver-dulness completely, and extending upwards sometimes almost to the clavicle and downwards for a greater or less distance into the abdomen. Over this area the *bell sound* may be obtained, and part of it will become dull when the patient assumes the erect posture. For these reasons, the name of *sub-diaphragmatic pneumothorax* has been applied to this condition.

---

[1] *Deutsche Archiv f. klin. Med.* xxxiv. p. 169.

In both forms the heart may be very much displaced. Sometimes the contents are creamy, inodorous pus, or the abscess may contain the chocolate-coloured pus which is characteristic of hepatic abscess; if the matter have some communication with the bowel, it is sure to be highly offensive. An abscess caused by rupture in the upper part of the stomach is much more difficult to diagnose. It will probably be confined by adhesions along the margin of the ribs, and the matter may be situated far back near the spine: it may or may not extend between the liver or spleen and the diaphragm. The proper course to pursue in dealing with this condition may be illustrated by a case which occurred in the Brompton Hospital. It was that of a young woman who had previously exhibited some symptoms of dyspepsia. A needle inserted through the ninth interspace in the line of the angle of the scapula had withdrawn some highly offensive pus, it was supposed from the pleura, and I was told it was a case of empyema. My own exploration showed the presence of clear inodorous fluid in the pleura. Suspecting the nature of the case, I opened the abdomen below the costal margin at the top of the linea semilunaris, and ascertained the presence of adhesions. This wound was closed, and on a subsequent occasion I removed a part of the ninth rib in the mid-axillary line, reflected the pleura from the diaphragm, cut through the latter, and then introduced my finger along the anterior border of the spleen, between the stomach and the diaphragm, until I reached the abscess somewhere near the spine. The patient did well. I should add, however, that I have opened one of these abscesses without recognising its true nature. A sub-diaphragmatic abscess caused by rupture of an abscess of the liver is most conveniently opened after removing a piece of rib in the mid-axillary line below the level of the pleura; one that starts from ruptured ulcer in the duodenum or transverse colon should be opened below the ribs.

A suppurating hydatid of the liver may also lead to confusion in diagnosis. The presence of an obvious and characteristic tumour in the abdomen will probably prevent a mistake, but it must be remembered that the tumour is not always present, and that, if strong adhesions have taken place between the liver and the diaphragm, very extensive dulness may be produced upwards, with little or no apparent enlargement downwards of the viscus. I was once asked to deal with a case of supposed empyema in which the tube had been inserted through the ninth interspace in the scapular line; dulness extended to the third rib in front, and passed across the apex of the axilla, becoming less definite as it reached the back; there was great displacement of the heart, and an indefinite rounded swelling was to be felt in the abdomen. On enlarging the original opening, I found that the matter was contained in a vast sub-diaphragmatic hydatid. If there had been any doubt about the nature of the case, it was cleared up subsequently by the fact that large quantities of bile escaped through the wound for some time, causing an extraordinary degree of emaciation, with consequent bed-sores, from which, however, the patient rapidly recovered on the sudden closure of the ruptured bileduct.

Pyopericardium.—Pus in the pericardium may distend that cavity to an enormous size, displacing the lung to a corresponding extent. The *symptoms* of pyopericardium may be distinct, or they may be very ambiguous. Pyopericardium is not unfrequently associated with empyema, and the very ambiguity of the symptoms may lead to the suspicion that the latter, not the former, is the cause of the physical signs that are met with. It thus happens that a correct diagnosis has been made only as the result of an incision intended to open an empyema. The sort of confusion that may arise may be illustrated by two cases. One was that of a young man who had extensive dulness on the left side of his chest, with other symptoms of the presence of fluid; he was also expectorating large quantities of pus. A quantity of pus was let out of the pleura, and on introducing the finger a large, soft, rounded body was felt, which was taken to be the semi-solidified lung. The patient improved for a time, but his expectoration never stopped, and at last he died, when it was discovered that what had been taken for the lung was really the pericardium tightly distended with pus. Another case is that of a little girl, whose obvious trouble was a left empyema, the evacuation of which was followed by numerous emboli in the systemic vessels, causing gangrene of both legs, albuminuria, and sloughing of the upper half of the spleen. She lived six weeks, and after death it was found that her primary trouble was an abscess in the muscular substance of the heart, followed by pyopericardium, which had no doubt given rise to the empyema.

Should the distended pericardium be felt, as in the first case, there can be no objection to opening it by an incision on its outer side; but it need not be pointed out how difficult this condition is to recognise, nor with what serious consequences a mistake might be followed. If the case be recognised as one of uncomplicated pyopericardium, probably the wisest situation for the incision is the fifth left interspace close to the sternum. The pleura will probably have been obliterated by adhesions, but it should not be assumed that this is the case. The incision should therefore be made carefully in the intercostal space, and, if a healthy pleura is met with, it should be turned aside, the removal of rib cartilage being avoided if possible. If sufficient room is not obtained without doing so, it is better to take away a little from the cartilage above and the cartilage below than to cut any one cartilage across. The subsequent treatment of pyopericardium does not come within the scope of this treatise, but it may be mentioned in passing that, if aspiration of the pericardium is required, the seat of election for the puncture is the point referred to, though some have recommended one in the intercostal space above and some the fifth space on the right side of the sternum. A reference to the description of the anatomy of the pleura (pp. 26, 27) will show that in no case is it certain that the pleura will be avoided.

R. J. G.

CHAPTER LV

PERIPLEURITIS, CARIES AND NECROSIS OF RIBS, MEDIASTINITIS

	PAGE
Peripleuritis	621
Acute	621
Chronic	622
Caries and necrosis of ribs, &c.	623
Mediastinitis	624

In the year 1861 attention was drawn by Wunderlich[1] to a condition which he called **peripleuritis** -that is to say, inflammation of the tissue between the pleura and the chest walls—but long before that it was recognised, and indeed it is said to have been described as long ago as 1679 by Bonet. Since 1861 the subject has been much discussed, principally in Germany, some disapproving of the term and asserting that the definition is without pathological interest or clinical importance. The description is made to include two totally different conditions, one of which, if it exists at all, must be exceedingly rare, while the other is recognised by all practical surgeons.

Acute form.—Of this, the rare form of the disease, I have no personal experience, and therefore borrow a description from Professor Riedinger's account of it in the Deutsche Chirurgie.[2] He says that it mostly begins with a rigor, and is followed by pain on the affected side; the respiration is superficial, the movement of the affected side is impaired, and there may be great dyspnœa; there is limited dulness; vesicular breathing and vocal fremitus are either diminished or absent, and, as pus forms and increases in quantity, a tumour develops on the surface of the thorax, which is increased by the act of expiration, and which exhibits more or less distinct fluctuation. The swelling is painful, and ultimately becomes reddened and œdematous. The affected intercostal spaces are widened, but above and below the ribs are approximated. There are the usual signs of fever, and there is generally cough, with or without expectoration. Nephritis and albuminuria are said to be common accompaniments. These abscesses all have a tendency to open externally. This description is evidently intended to apply to some acute inflammatory process analogous to infective cellulitis. I can only repeat that I have not met with this condition independent of injury or cellulitis elsewhere.

---

[1] *Archiv der Heilkunde*, ii. 1861, p. 17.

[2] *Verletzungen und chirurgische Krankheiten des Thorax und seines Inhaltes* Lieferung 42, Stuttgart, 1888.

The more *chronic form* of the disease is, on the other hand, frequently met with; it does not arise spontaneously, but depends upon some cause of suppuration external to the pleura. By far the commonest is caries, or necrosis of the ribs, rib-cartilages, or sternum; a less common cause is caries of the dorsal or cervical vertebræ. Caries of the vertebræ, as has been said, may give rise to a genuine empyema. I have seen at a post-mortem examination the lower part of an abscess starting from the body of a cervical vertebra bulging into the apex of the pleura and apparently on the point of bursting into it; but much more frequently it results in an abscess which tracks along the ribs, separating the pleura from them till at last it points through one or other intercostal space. The physical signs produced by such a collection of matter, if it be at all large, simulate very closely those of an empyema; and, after the cavity is opened and the finger is introduced, it is often by no means clear whether it be external to the pleura or not. Frequently also it happens that the rib along which the abscess has burrowed has been denuded of its periosteum, a condition which still further confuses the diagnosis; and, unless the surgeon's attention be particularly directed to the spine, he may well fail to detect the signs of caries, which are probably much less striking than those in the chest. When what is apparently a small empyema in the back continues to discharge indefinitely, it is better always to ask oneself whether it is possible to exclude the existence of spinal caries. I have seen many cases in which the diagnosis was doubtful, and a good many in which the actual state of the case was only discovered at a post-mortem examination. As an example may be mentioned the case of a young officer whose trouble began with right pleurisy, followed by sciatica first on the left and then on the right side; after a while abscesses developed in the back; he was seen by many surgeons, some of whom thought it was a case of empyema, some of caries of the rib, and some of spinal caries, with which opinion I agreed, although none of the usual signs of Pott's disease were present. After four years of invalidism, during which the sinuses never completely closed, he rapidly developed two large abscesses in the buttock, which had evidently made their way through the great sacro-sciatic foramina. These were opened and quickly healed, leaving him strong and well, but with a stiff dorsal spine. He returned to his duty and remained in good health for nearly three years, when, after an attack of influenza, a large collection of matter formed inside the thoracic wall on the right side, which from the very beginning of the case had remained more or less dull. This I opened in two places on two separate occasions, but, although I explored the cavity with the utmost care, I am unable even now to say whether the very dense structure which formed its inner wall was the outer surface of the costal or of the visceral pleura. Had I seen the case then for the first time I should have had no doubt that it was one of empyema. As it is, it seems to me quite possible that it may have been fresh suppuration in connection with the caries of the spine, a view that is strengthened by the fact that, after months of very serious illness, the wound still remains unhealed.

Caries and necrosis of ribs, &c.—Caries of the ribs, rib cartilages, and sternum, like caries of the spine, is mostly tubercular. It is almost always very chronic, and it is met with frequently in patients who are subjects of phthisis, but is by no means an uncommon manifestation of senile tuberculosis. Necrosis of the ribs may arise from the same cause, and then is often associated with caries. It may also arise from injury, or from acute infective osteomyelitis. Except in the latter case, both caries and necrosis are usually chronic, the patient's attention being first directed to the spot by a little dull pain, or perhaps by the appearance of a swelling. The changes in the bone may consist of caries on the inner surface, or on the outer surface, or sometimes of one or more perforations through the shaft of the rib, or it may be a central necrosis, or a superficial necrosis on either aspect. Caries or necrosis of the sternum may also be central, or on the anterior or posterior aspect. Caries of the articulation between the manubrium and the gladiolus, or in the chondrosternal articulations, does not differ in character from similar disease elsewhere. The situation in which the mischief occurs determines the seat of the abscess. Thus if it be on the outer surface of the rib or sternum, the abscess will probably be completely, or almost completely, superficial; if it be on the deep aspect of either bone, it will in the first place separate the pleura from the ribs, and ultimately make its way through an intercostal space to the surface. In cases of central necrosis, or perforating caries, or disease of the articulations, it is probable that matter will collect on both surfaces of the bone or cartilage from the very first. Superficial cases give rise to no difficulty in diagnosis. The others may be easily mistaken for empyemata, especially if, as is sometimes the case, the peripleuritic collection of pus is extensive. In all except the superficial abscesses an impulse may sometimes be obtained on coughing. As in the case of caries of the spine, the abscess often follows the rib for a considerable distance from the seat of mischief before pointing. Below the level of the pleura it may burrow extensively amongst the fibres of origin of the diaphragm, pointing as it does so through several intercostal spaces.

An abscess connected with the posterior surface of the sternum may point through an intercostal space or at the episternal notch, or at the epigastrium, or it may come to the surface by perforating the bone. It is impossible to refer to these cases without pointing out how often the hard tumour which results from an abscess in connection with the rib in its earliest stages is mistaken for a malignant growth or a gumma.

The *treatment* of these cases depends upon the situation of the disease. Unless the affected rib be in front of the pericardium or in some very inaccessible region—such, for example, as that of the first rib—it is wisest to remove the whole of the diseased part, unless it prove to be an example of necrosis with a loose sequestrum, under which circumstances it may be dealt with upon the same principles that guide us in treating a similar condition in the long bones. The most satisfactory results after removal of a piece of rib are usually obtained by leaving the wound open and dressing it from the bottom. But occasionally it is best to sew the wound up completely, and hope for union by first intention—a hope which is occasionally not disappointed.

Scraping of caries of a rib is not a very satisfactory procedure; the patient, it has been said, is probably tubercular, and a recurrence of the mischief is very probable. I have seen the operation followed within a few days by acute tuberculosis. Caries of the joint between the manubrium and the gladiolus involves the application of a sharp spoon to this articulation. The immediate result may be the complete separation of the two parts of the sternum, with the consequent increase of movement referred to on page 9. This may, especially in the case of a child, interfere considerably with respiration. In an adult it will probably cause no inconvenience, and it will be followed, if closure of the wound takes place, by anchylosis of the two parts of the bone. Poststernal abscess is very difficult to deal with. It has been reached by trephining the bone, but usually it is necessary to wait till it points in one of the situations mentioned above.

Mediastinitis.—Closely allied to these peripleural inflammations is the disease known as *mediastinitis*, or inflammation of the mediastinum. The poststernal abscesses, indeed, described above, really come into this category, but there are other conditions depending upon inflammation of lymphatic glands and caries of the spine which give rise to chronic abscess in the mediastinum, and there is also an acute mediastinitis analogous to that which was referred to under the name of acute peripleuritis. It is, however, though rare, a much better known complaint than the latter, and arises most frequently in connection either with septic processes starting in the neck, such for example as *angina Ludovici*, or inflammations following cut throat, tracheotomy, or the removal of thyroid or other tumours, or it may be caused by wounds of the œsophagus, or perforation of the œsophagus or trachea. All abscesses in the mediastinum are liable to give rise to much pain and to dyspnœa. The acute form is accompanied by the usual signs of septic poisoning. The chronic forms may be the cause of some of the most puzzling symptoms with which the physician or surgeon has to deal. Not unfrequently does it happen that a series of these anomalous symptoms has been cleared up by the expectoration of a quantity of pus caused by the bursting of a suppurating gland into the trachea or into a bronchus, a *dénouement* which has not unfrequently proved fatal. The following case will illustrate the sort of difficulties which may be met with. A young officer complained first of a right brachial neuralgia; he then developed pleurisy with effusion which became purulent on the left side. At this time it was thought that he might have a mediastinal abscess. After prolonged treatment the empyema closed, and the patient apparently recovered, but only to die years afterwards of profuse hæmoptysis caused by laceration of the arch of the aorta by a calcareous mediastinal gland. This case was under the care of Dr. Sydney Phillips, with whom I saw the patient. It is fully described by him in the 'Medico-Chirurgical Transactions' for 1897.

It may sometimes be justifiable to search for an abscess in the anterior mediastinum by trephining the bone, but any one who has done so and passed his finger along the front of the heart to the great vessels will appreciate more vividly than is possible from mere anatomical knowledge how extremely dangerous the situation is for surgical interference, and any such operation in the posterior mediastinum would, I think, be beyond the limits of legitimate surgery. R. J. G.

NOTE.--Inflammation of the mediastinum may be set up by an aneurysm of the arch of the aorta, and subsequently the contraction of the inflammatory products may lead to complete obstruction of the superior vena cava. This was proved on autopsy to have occurred in a case under the care of the writer. Dr. G. B. Perez[1] has drawn attention to a physical sign which is present in some cases of mediastinitis, viz. a crackling or crepitant sound audible over the sternum and elsewhere when the arms are raised above the head, and again when they are lowered to the side. Dr. Perez kindly allowed the writer to examine the case in which this sign was first observed. The patient was a lady who had suffered on several occasions from attacks of 'mediastino-pericarditis.' On raising the left arm above the head, whilst the patient was in the recumbent position, crackling sounds were heard over the whole length of the sternum, at the supra-sternal notch, along each side of the sternum, over the sterno-clavicular articulations, and also along both clavicles. Raising and lowering the right arm also gave rise to a similar sound, but it was then less distinct. The occurrence of such sounds in cases in which changes were probably present in the mediastinum has since been noticed by other observers. It is not unlikely that, as suggested by Dr. Harris, these sounds are produced by a slight degree of movement of the large vessels and of the arch of the aorta taking place when the arms are moved in the manner described; this would drag upon any adhesions which might be situated around the vessels external to the pericardium and stretch them to some extent. J. K. F.

---

[1] *Brit. Med. Jour.*, vol. ii., 1896, p. 717.

CHAPTER LVI

PNEUMOTHORAX

PAGE
Etiology 626
    Causes acting from without 626
    From disease within the chest 627
        (*a*) Of the lungs and bronchi 627
        (*b*) Of other organs 628
    From decomposition 628
Morbid anatomy 628
Pathology 629
    Process of re-expansion and of absorption 630
        Rate of absorption 630
        Composition of the contained air 630
Pneumothorax in cases of pulmonary tuberculosis 630
    Frequency 630
    Side affected 631
    Site 631
    Number of perforations 631
    Size of perforations 631
    Nature and amount of the effusion 631
    Type of the disease 632
    Age 633
Symptoms 633
Physical signs 636
    Intra-thoracic pressure. 638
Course and terminations 638
Diagnosis 639
Prognosis 639
Treatment 641

ETIOLOGY

THE conditions under which air may gain access to the cavity of the pleura may be divided primarily into (*a*) those which arise external to the thorax, and (*b*) those which originate from disease of the organs contained within the chest; (*c*) the spontaneous decomposition of a pleural effusion.

Pneumothorax from without.—The occurrence of pneumothorax from *traumatic conditions* is considered in the chapters on Injuries of the Lungs and Injuries of the Pleura (*vide* p. 494 and 536). It may be repeated here that perforation of the chest wall and lung, or of the lung only, by a fractured rib are conditions not necessarily or indeed commonly attended with pneumothorax, owing to the cohesion of the opposed pleural surfaces and because the track of the wound in the lung may be closed by effusion of blood.

An *abscess* of the chest wall may open both externally and into the pleura and thus give rise to pneumothorax.

Perforation of the pleura by an abscess in communication with an air-containing organ in the abdomen is a rare cause of pneumothorax. In a considerable proportion of the cases arising in this way, the primary lesion is an ulcer of the stomach.

Pneumothorax from disease within the chest.

(*a*) *Diseases of the lungs and bronchi.*—Probably in about 90 per cent. of all cases the condition results from the perforation of the pleura by a tubercular process affecting the subjacent lung. In the remaining 10 per cent. perforation of the lung by an empyema comes first in point of frequency amongst the causes, and then pulmonary gangrene. Amongst still rarer causes abscess and hydatids of the lung or mediastinum, necrosis of the pleura over a septic infarction, and the rupture of an emphysematous vesicle may be mentioned.

By some writers the occurrence of pneumothorax from the latter cause has been doubted; but cases of the kind, although extremely rare, have certainly been observed.

The following case may be cited:

A middle-aged female was brought in dead to the Middlesex Hospital on November 10, 1889. Death had occurred shortly after labour.

P. M.—'There was old collapse of the left lung, which was "bound down" by adhesions. On the right side there was recent pneumothorax producing complete collapse of the lower lobe and partial collapse of the upper and middle lobes, which were fixed to the chest wall by fibrous adhesions. There were numerous emphysematous bullæ along the anterior margin of the right upper lobe, one of which had ruptured and caused the escape of air into the pleural cavity.'

The only available respiratory organ had thus been rendered functionless.

The occurrence of pneumothorax in cases of whooping cough is probably due to a similar cause, but the condition of the lung is then more often one of extreme hyper-distension than of true emphysema.

In the chapter on Interstitial Emphysema (*vide* p. 181) several cases are described in which pneumothorax was present after tracheotomy for laryngeal diphtheria, and one in which tracheotomy had not been performed.

Ulcerative bronchiectasis, and the changes resulting from the presence of a foreign body in a bronchus, may also be mentioned as rare causes of the condition.

Pneumothorax occasionally occurs as a complication of typhoid (*b*) *Diseases of other organs.*—Cancer of the œsophagus may rupture into the pleura and produce pneumothorax.

Cases are recorded in which a communication has been established between the lung and pleura by the rupture of a bronchial gland in both directions.

Pneumothorax from decomposition.—The occurrence of pneumothorax from the spontaneous evolution of gas from a putrid pleural effusion must be extremely rare. Some writers deny the possibility of such a condition arising, others admit it, but absolute proof is wanting. In some cases apparently of this nature, it is highly probable that perforation of the pleura had previously taken place and the opening had become closed.

Occurrence of pneumothorax in apparent health. It will be convenient to consider here the occurrence of pneumothorax in persons apparently healthy. The late Sir William Gull is reported to have said, 'Call no man healthy until he is dead and Dr. —— has made the post-mortem.' Pneumothorax sometimes occurs in patients apparently in perfect health; these cases, as a rule, recover, but if one should chance to die it is probable that a post-mortem examination would prove him to have been, whilst apparently healthy, really the subject of a limited area of tubercular disease of the lungs, or perhaps of emphysema. In the majority of tubercular cases, as pointed out by Dr. S. West, when pneumothorax occurs the patients are not engaged at the time in any violent respiratory effort; in some, indeed, the lesion occurs during sleep. It is possible, however, by violent expiratory effort to produce a force capable of rupturing the lung; but, as will be seen on reference to the cases described in the chapter on Interstitial Emphysema (*vide* p. 181), mediastinal emphysema is the more common result.

MORBID ANATOMY

Care is necessary in opening the pleural cavity whilst making an autopsy on any case where the presence of pneumothorax is suspected. Fagge recommends that the tissues should be dissected off so as to form a pouch, which can be filled with water, or that water should be poured into the abdomen, and the diaphragm then perforated with a trocar. We have found that a simple way is to carefully dissect off the intercostal muscles and expose the pleura in one or more interspaces. If the parietal layer is not thickened, the visceral pleura can then be seen through it; if it is, the dissection should be continued and a small opening made. There is then no difficulty in observing whether the surfaces were previously in contact.

If the pressure within is positive the escape of the air is forcible, but this is not the most common condition.

On removal of the sternum and cartilages the lung may be found to be completely or partially collapsed, and the heart and mediastinum displaced.

The appearances of the pleura necessarily depend on the presence or absence of inflammatory changes and their nature. If the condition is merely a pneumothorax, the surface may present its normal glistening aspect; but, on the other hand, the walls of the cavity may present the appearances characteristic of acute inflammation or of an empyema.

After removal of the lungs careful search should be made for the site of the perforation, and if this is not readily found air should be forced through the main bronchus and the surface carefully watched to note where it escapes. If the pleura is covered with a thick layer of organised lymph, it may be impossible to discover the site of the opening. The fact, however, that in the sixty-five consecutive cases noted below it was found in all but four proves that the difficulty in so doing is not really very great.

PATHOLOGY

A knowledge of the physics of the chest is essential to an understanding of the pathology of pneumothorax, *i.e.* why it occurs under certain circumstances and does not under others. The normal elasticity of the healthy lungs is a retractile force tending to produce collapse, and is constantly being exerted on the chest wall, heart, and mediastinum; it is probably equal to about seven millimetres of mercury or 3·8 inches of water.

The heart may be therefore regarded as held in its normal position by two oppositely placed elastic bands of equal tension.

When air gains access to one pleural cavity the band on that side is severed, and the heart is invariably drawn over to the opposite side by the elastic traction of the other band. This explains why the displacement of the heart in pneumothorax is immediate and independent of the existence of any positive pleural pressure, although the extent of the displacement is affected by the presence or absence of that condition.

Dr. S. West has shown that the force of cohesion of the two serous surfaces of the pleura is considerably greater than that exerted by the elastic traction of the lungs, and that it may be estimated at 12·5 millimetres of mercury or 6·8 inches of water. The existence of this force, which may be experimentally demonstrated in a variety of ways, explains the absence of pneumothorax and of collapse of the lung in some cases of injury in which air has free access to the surface of the pleura exposed in the wound. The force continues in operation to its full extent only so long as a fine film of moisture intervenes between the surfaces. If dry, they do not cohere at all; if wet, the force of cohesion of any serous membrane is much diminished, and when air is present it disappears altogether. For the separation of the surfaces a force is required which will overcome that of cohesion, and this in the case of the pleura is supplied by expiration.

When separation has been effected the elasticity of the lung comes into play, and continues to act until the lung has been reduced to one-eighth of its normal volume (Donders). Air continues to enter the pleural cavity during inspiration until the pressure is equal to that of the atmosphere; after this is attained it can only enter during expiration, and great over-distension with air alone can only occur when the opening is valvular and violent expiratory acts, *e.g.* cough, constantly recur. When a positive pressure is present during inspiration it is in most cases due, as Dr. West points out, to the presence of fluid, but the absence of positive pressure on inspiration does not imply that no effusion has occurred. On expiration the pressure is always positive; if the opening is widely patent, it may be very slight; but, if closed or of minute size and the expiratory effort great, as in severe cough, the difference between the inspiratory and expiratory pressure is considerable.

Process of re-expansion and of absorption.—The exact manner by which the re-expansion of the lung is brought about is a question of extreme difficulty. It is intimately connected with that of the mode of absorption of fluid and air from the pleural cavity. Gases are removed by simple absorption and diffusion, or the process is one of absorption aided by chemical combination. The mode of absorption of fluid is described in the chapter on Pleurisy. The matter is fully considered by Dr. S. West in the Bradshaw Lecture on Pneumothorax, to which we must refer our readers for more detail, as the subject, although of much interest, is not one of great practical moment. The *rate of absorption* of air may be extremely rapid. As stated in the section on treatment, a quantity of air sufficient to give a tympanitic note all over one half of the chest may completely disappear in three days.

The composition of the air in the pleural cavity depends upon its mode of entrance, and upon the time it has been present there. In a case of punctured wound it is at first the atmospheric air; when it has gained access from the lungs it has the same composition as that in the alveoli: $N=79{\cdot}5$, $CO_2=4{\cdot}38$, $O=16{\cdot}0$. When the opening is closed and it has been long present the oxygen disappears and the carbonic acid and nitrogen increase in quantity.

PNEUMOTHORAX FROM PULMONARY TUBERCULOSIS

In such a very large proportion of cases the condition occurs as a complication of pulmonary tuberculosis that interest chiefly attaches to the lesions then met with.

Frequency.—The post-mortem records of 1,000 cases of tubercular disease of the lungs occurring at the Brompton Hospital for Consumption include sixty-five cases of pneumothorax, the analysis of which is taken as the basis of the description given below. This proportion (6·5 per cent.) accords fairly with the percentage given by other observers.[1]

---

[1] Douglas-Powell and West (five per cent.)

Side affected.—Most writers state that the left side is more frequently affected than the right; some even give the ratios as 2 : 1 or 3 : 2. In the above-mentioned cases the difference was much less, the left side being affected in thirty-four and the right in twenty-nine. In two the affection was bilateral.

Of eighty-three cases observed by West[1] the right side was involved in forty-one, and the left in forty-two.

The *site* of the perforation could not be found in four of the cases which we have analysed, and it is not stated in one, but including the bilateral cases and those in which more than one lobe was affected sixty-four perforations were thus distributed.

Right lung		Left lung	
Upper lobe	13	Upper lobe	21
Middle lobe	5	Lower lobe	14
Lower lobe	11		
	29		35

The mid-lateral aspect of the upper lobe was the most common site.

Number of perforations.—In twelve cases there was more than a single opening. In two they are described as 'numerous,' in one there were 'several,' in two four, and in six two. More than one lobe of a lung was perforated in three cases.

Size.—The opening was generally small and rounded, but may be of considerable size; in one case it was of the size of a shilling and presented bevelled edges.

The nature and amount of the effusion.—Of the sixty-five cases above mentioned the nature of the contents of the pneumothorax is given in all but one. In seven there was no effusion and no evidence of inflammation. In forty-three cases pus was present; this was either greenish yellow or dirty grey in colour, and in three of these it was fœtid. In four cases the effusion is described as turbid, blood-stained, or as flaky. In two it was sero-purulent, and in four it consisted of clear serous fluid. In one there was a gelatinous exudation with lymph flakes and in two there was recent lymph on the pleura without any effusion of fluid.

Of the two cases of double pneumothorax, in one the left cavity opened spontaneously in the third interspace in the mid-axillary line after paracentesis had been performed and 218 oz. of pus removed; the right pleura contained air and the membrane was merely roughened. In the other case the left cavity contained 20 oz. of pus, which, as the history showed, had formed in six days, whilst in the right cavity there was air only.

In only five cases is there a definite statement that tubercle of the pleura was present.

The history of the cases in which repeated tapping had been performed indicates that in the majority the effusion is first serous, then sero-purulent, and finally purulent. It may, however, be purulent from the onset or remain serous for a considerable period.

The greatest amount of effusion found was 4½ pints; it is described as gelatinous-looking fluid containing flakes of lymph. The clinical history of this case shows that the onset of the pneumothorax was five weeks before death. There is, however, no direct relation between the duration of the condition and the amount of fluid effused.

The type of the tubercular affection.—In the majority of cases the disease is of the caseous type, and perforation occurs owing to the fact that there has not been sufficient time for the formation of pleural adhesions. In others, after a period during which the course is more or less chronic, perforation occurs whilst a rapidly extending tuberculosis is in progress. In a few the disease has run a chronic course throughout.

As a rule, extensive lesions are present in both lungs, but pneumothorax may supervene when the area affected is limited, and in such cases it is due to the accidental softening of a nodule situated just beneath the pleura. When the patient has lived some time after the occurrence, the tubercular lesions in the lung of the affected side nearly always give the impression of not having made much advance of late, whereas on the opposite side they may present signs of marked activity. We have never seen appearances which pointed to the conclusion that acute extension of tubercle had occurred in a lung after complete collapse.

It would indeed be strange if tubercular disease could make rapid progress in a lung which is bloodless and airless, and in which, therefore, all the ordinary channels through which the virus of the disease is spread, viz. the bronchi, vessels, and lymphatics, are more or less obstructed.

The view here stated that complete collapse of the lung following pneumothorax with pleural effusion may modify or arrest the progress of tubercular lesions has hitherto been generally held, but it is not in accordance with the recently expressed opinions of some physicians. Our own opinion is, however, founded on some pathological experience, and so far we see no reason to alter it. It may, however, be well to cite briefly a classical case in support of this old-fashioned view. It will be found in a paper by Dr. H. M. Hughes, in the Guy's Hospital Reports,[1] on cases of pneumothorax with remarks.

'Miss P., æt. 26, had lost one brother from tuberculosis. A year ago had an attack of hæmoptysis followed by cough with expectoration. When first seen by Dr. Chalmers of Croydon, the expectoration was blood-streaked, there was pain in the right side of the chest, and urgent dyspnœa on exertion. The physical signs pointed to a lesion at the right apex. Attacks of pleurisy on the right side subsequently occurred.'

---

[1] *London Medical Gazette*, 1844, and *Guy's Hospital Reports*, vol. viii. second series.

Three months later she was found by Dr. Hughes to have right-sided pneumothorax with succussion splash. There were then no signs of disease in the left lung.

Five months subsequently paracentesis on the right side was performed, and three pints of turbid fluid removed, and the operation was repeated after another interval of two months. Recovery followed, with partial re-expansion of the right lung. She died eighteen months after the discovery of the pneumothorax.

P. M.**—The left pleura was firmly adherent. The left upper lobe was softening throughout, and caseous tubercles were present in the lower lobe. The right pleura contained 80 oz. of thick sero-purulent fluid.** ***Calcified tubercle*** **was found in the right upper lobe, and a cavity containing a** ***white chalk-like substance*** **separated from the surface only by thickened pleura.**

The **age** at which the lesion occurs most frequently is from twenty-five to thirty-five years—that is, when the mortality from tubercle is greatest.

SYMPTOMS

In a large majority of cases the onset is sudden and is accompanied by severe pain in the upper part of the chest or back; a sensation of 'something having given way,' or of air having escaped, is sometimes experienced, and the patient may be aware that the heart has changed its position.

The shock may be severe and the heart may temporarily fail; if so, the extremities become cold and a clammy perspiration breaks out. Dyspnœa, as a rule, appears quickly and may be intense. The patient suffers from great mental distress and an agonising feeling of want of breath. The pulse and respiration are markedly frequent. If in bed, he sits up and leans forward, the alæ nasi are widely dilated, the face may be livid or cyanosed, and the voice reduced to a whisper. The severity of the early symptoms depends chiefly upon the previous functional activity of the lung on the affected side. If this has already been greatly impaired by disease, its loss may be hardly felt and the onset will be latent; whereas, if it was the mainstay of respiration, death must quickly follow upon its collapse. Between these extremes there are many intermediate grades of severity.

The rapidity with which the air escapes into the pleural cavity is another factor in determining the urgency of the early symptoms. The slower this is, the greater is the time given to the other lung to accommodate itself to the new condition and the less severe the dyspnœa. Another reason why the onset is often but little marked in cases where there is advanced destructive disease of the lungs and general feebleness is that the respiratory needs of the blood under such circumstances are very small. It is quite common in cases of this kind in hospital for pneumothorax to be discovered during the ordinary routine examination of the chest, and without any very decided change having occurred to suggest its presence. In such cases pain is undoubtedly the symptom which is most rarely absent, and patients with rapidly advancing tubercular disease should be questioned upon this point at every visit.

The effect upon the temperature depends upon its previous range and the vitality of the patient. If perforation occurs whilst the morbid process is active and the pyrexia continuous and ranging high, a sudden fall of 5° or even 6° or more is generally observed; a subnormal temperature of 95° being often recorded in such cases.

If, however, the condition of vitality is low, or pneumothorax attacks a person 'apparently' healthy, the fall will be less.

As the shock passes off and recovery takes place, the temperature will rise quickly, nearly to its previous height in pyrexial cases. In other cases the course of the temperature depends upon the changes which take place in the pleura. If no inflammation ensues and the air is rapidly absorbed, pyrexia may be absent; on the other hand, if inflammation occurs, the subsequent course of the fever is of an irregular hectic type with recurring remissions.

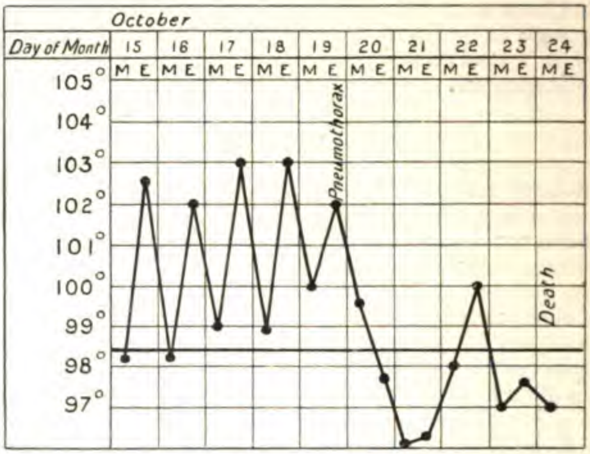


Fig. 145.—Temperature Chart from a Case of Pulmonary Tuberculosis in which Pneumothorax was accompanied by a Rapid Fall of Temperature

The temperature taken in the axilla on the affected side may for a time be lower than that on the healthy side. (Peters, Williams.)

In the majority of cases the extremely acute symptoms, which are due to shock and to the sudden reduction of the respiratory area and the displacement of the heart, pass off after a few hours, but the breathing continues rapid and may even increase in frequency as the air accumulates in the pleura. There is, however, no longer the extreme subjective sense of dyspnœa. Walshe, for example, mentions a case in which, although the respirations numbered 52 per minute, the patient was not conscious of suffering from any difficulty of breathing.

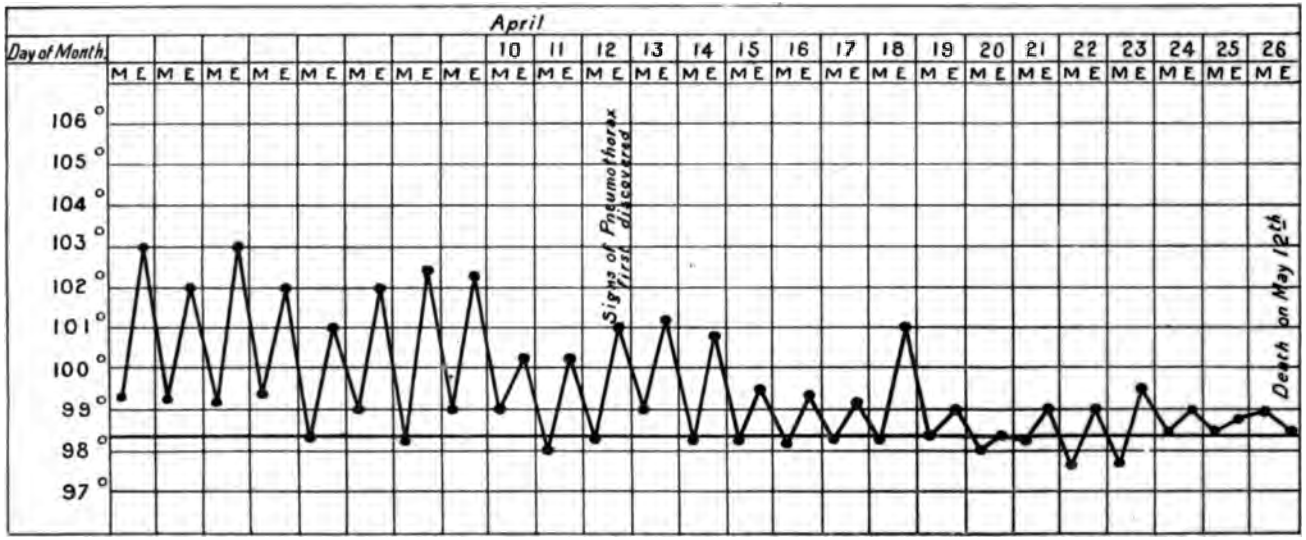


FIG. 146.—TEMPERATURE CHART FROM A CASE OF PULMONARY TUBERCULOSIS IN WHICH PNEUMOTHORAX OCCURRED WITHOUT MARKED SYMPTOMS

PHYSICAL SIGNS

Inspection.—The affected side after a time becomes motionless, and it may be obviously enlarged. The surface then appears smooth from the absence of intercostal depression and the shoulder is raised. The heart is displaced, and its impulse may or may not be visible. If positive intra-thoracic pressure is present, bulging is marked and the superficial veins may be distended. In pneumothorax due to the rupture of an empyema into the lung or externally, also in some cases secondary to abdominal disease, and generally where the perforation is large and continuously patent, there may not only be no increase in the circumference of the side, but it may be diminished, whilst in empyema of long standing retraction is almost invariably present, even if there be air in the pleura.

If the pneumothorax is circumscribed and the lung is held in contact with the chest wall over a certain area by previously formed adhesions, a limited amount of expansion of the affected side will still be observed. The expansion of the opposite side of the chest is increased after a time, but the intercostal spaces recede during inspiration.

Palpation.—Vocal fremitus is absent over the affected side unless thick bands of adhesion are present, when it may be to some extent retained. The exact site of the cardiac impulse and of the lower margin of the liver should be determined. It may here be again mentioned that the displacement of the heart occurs immediately the air enters the pleural sac. The liver will be displaced downwards in right-sided pneumothorax and with positive intrathoracic pressure possibly to such an extent that it is nearly on a level with the iliac crest. The spleen may also be displaced.

Percussion. *Air only present.*—The note is tympanitic over the air-containing cavity, and, owing to the displacement of the heart and mediastinum, this note may be obtained beyond the middle line of the sternum. The tympanitic note may be absent over the area occupied by the compressed lung, but Wilson Fox[1] states that he had observed it to be present in pneumothorax over an area where a consolidated lung was adherent, and also on the opposite side, over a consolidated and adherent apex. It is generally stated that the note differs in pitch according to the amount and the tension of the contained air, and that as the pressure increases the percussion note loses its tympanitic character and becomes muffled or dull. Dr. S. West[2] states that he has never observed this change, and our own observations are in accordance with his.

Percussion in the lower axillary region on the right side may confirm the observation that the diaphragm is displaced downwards; on the left side the normal stomach note in that region interferes with this observation. An area of dulness corresponding to the site of the displaced heart may be mapped out, but the heart may be overlapped owing to the compensatory enlargement of the sound lung. The precordial dulness is quite obscured in left-sided pneumothorax.

---

[1] *Diseases of the Lungs and Pleura*, p. 1110. [2] *Lancet*, vol. ii. 1887, p. 358.

It is stated that when the fistula is open, a 'cracked-pot sound' may be produced, and that the note varies in pitch according as the mouth is open (low) or closed (high). We have not verified this observation.

On combined auscultation and percussion over a pneumothorax or any large air-containing cavity a note of a metallic character is audible.

Air and fluid.—Where effusion occurs, the fluid collects at the base of the cavity, and on percussion a dull note is elicited. It is to be remembered that, owing to the flaccid condition of the diaphragm, which may bulge downwards towards the abdomen, a considerable amount may collect without producing dulness at the levels at which it ordinarily occurs in pleural effusions. In the left lower axillary region, and in the nipple line below the sixth rib, the dulness replacing the stomach note proves the presence of fluid and the displacement of the diaphragm downwards. The level of the dulness on light percussion can be shown to be horizontal, and it shifts according to the position of the patient. The percussion note above the level of the fluid retains its tympanitic quality, but if the lung does not re-expand and the air is gradually replaced by fluid, the whole of the side becomes dull.

Intercostal fluctuation and a thrill may be felt on percussion. The latter is said by Walshe to be most marked in the area intermediate between that of tympanitic resonance and that of the dulness due to the presence of fluid.

Auscultation. *Air alone.*—The vesicular breath sound is absent, but over the site of the collapsed lung a distant tubular sound may be present. Loud amphoric breathing may be heard; this is most marked when the entry and exit of air are free, but it may be present when the opening is closed, the peculiar quality being then due to the passage of bronchial sounds through a large resounding cavity. The voice sounds, cough, and any adventitious sounds also acquire an amphoric quality. Metallic tinkling or echo is often heard, either with respiration, the voice, cough or movement. It may also be induced by the act of swallowing, and by the movements of the heart or by pericardial friction.

The bell sound, or *bruit d'airain*, is produced by pressing a coin flat upon the part of the chest corresponding to the pneumothorax, and striking it with another coin whilst the ear or stethoscope is applied to the opposite aspect of the chest or elsewhere over the cavity.

The ringing quality of the note varies much in different cases.

Air and fluid.—All the signs just described may be observed, but, in addition, the Hippocratic succussion splash may be obtained. This is demonstrated by shaking the patient whilst the ear is applied to the chest, or he may be able to render it audible to the bystanders by shaking himself. The shock may also be felt by the hand placed upon the chest.

A displacement murmur may possibly be present, but it is very rarely observed; we have never met with a case of the kind. It probably only occurs when the pressure in the cavity is extreme.

Mensuration.—A cyrtometric tracing of the chest will more exactly demonstrate the degree of enlargement; it may possibly prove either that there is no enlargement or that the side is retracted.

Intra-thoracic pressure.—If it is desired to ascertain the state of tension within the cavity an aspirator needle must be inserted and connected with a mercury manometer. The mercury will then be observed to rise and fall with respiration, the difference between the pressure on inspiration and expiration being termed the 'respiratory oscillation.' In health this is estimated at about 3 mm. of mercury, or about an inch and a half of water, and in pneumothorax it is often of equal range; but with a widely patent opening, or when the perforation has closed and the tension is high, it may be less than the normal (West). When the opening is valvular, air continues to enter the cavity only so long as the pressure inside the pleura during the deepest inspiration is less than that of the atmosphere. On expiration the valve closes and the air cannot escape. When equilibrium is established the entry of air ceases. A violent expiratory effort, such as a cough, affords the only means by which a positive intra-thoracic pressure can be established with air alone; but an effusion of fluid may convert a pressure of zero into a positive pressure.

COURSE AND TERMINATIONS

The progress of a case depends greatly upon the condition of the patient preceding the onset of the affection, and upon the subsequent changes in the pleura. In traumatic cases and in pneumothorax affecting an 'apparently healthy' person, as already stated, the air may be quickly absorbed and recovery may be rapid.

If pleurisy supervenes, it implies that, in addition to the air, infective organisms have entered the pleural cavity. These may be streptococcus pyogenes aureus, staphylococcus and saprogenic organisms, or the bacillus of tubercle, and the rapidity with which the inflammation progresses depends upon the virulence of the organism.

It is interesting to note that amongst the sixty-five cases examined post-mortem, there is a definite mention of tubercular changes in only five. Unfortunately it is not expressly stated that they were absent in the other cases, and we think that more careful bacteriological examination of the effusion and microscopical examination of the pleura is required before the exact cause of the pleurisy can be determined. When it follows quickly on the perforation it is most probably of septic and not of tubercular origin.

This is certainly the case in pneumothorax following pulmonary gangrene or abscess, and in such cases the inflammatory process is extremely acute.

DIAGNOSIS

It may be very difficult to recognise a pneumothorax limited to the base and due to the rupture of an emphysematous vesicle, the heart being retained in position by adhesions; partly, no doubt, because it is an extremely rare condition, and the probability of its occurrence may not be present to the mind. We have observed a case which proved on post-mortem examination to be of this nature. The diagnosis made was pulmonary collapse; the breath sounds were absent, and the percussion note was uniformly tympanitic, owing to emphysema of the neighbouring lung.

The diagnosis from emphysema is considered in the chapter on that subject (*vide* p. 175).

When the lung is completely excavated so that nothing but the pleural capsule remains, the condition closely simulates a pneumothorax. Amphoric breathing, metallic tinkling, and the bell sound may be present; the two former signs are common, the latter is rare; but we have carefully noted its occurrence. A succussion splash was heard in a case of this kind by Laennec. In one case we were able to diagnose the co-existence of pneumothorax at the right apex, and a large cavity at the base by the distinct limitation of the amphoric breathing to the latter area and the absence of breath sounds above.

The position of the cardiac apex in this, as in so many other conditions, generally determines the diagnosis. With excavation of the lung it is more likely to be displaced to the affected than to the sound side, the chest wall is generally retracted, and the vocal fremitus will not be absent. If the position of the cardiac impulse is unaltered the physical signs should be very clear before a diagnosis of pneumothorax is made.

In asthma the extreme prolongation of the expiratory act and the presence of general wheezing are distinctive.

The diagnosis from sub-diaphragmatic abscess is considered in the chapter on that subject (*vide* p. 617).

PROGNOSIS

The disease occurs under such a variety of conditions that statistical statements are of little value, unless the cases are carefully grouped. As already stated, when the affection occurs in persons not obviously out of health the prognosis is favourable, as pleurisy rarely follows, and the air is rapidly absorbed. The probability of the presence of pulmonary tuberculosis in such cases must be borne in mind.

Speaking generally, if only air is effused, and the patient recovers front of the chest; yet in three days it may be found to have completely disappeared. In these cases, therefore, no treatment is required.

When, however, pneumothorax results from the pumping out of air through a valvular opening in the lung, the accumulation in the pleura rapidly increases, and the lung becomes collapsed and compressed; the opposite lung is pressed upon in the same way, and the heart is pushed over to the opposite side; the dyspnœa then becomes extreme, and, unless relief be afforded, the patient will die. Relief is to be obtained by passing a small trocar and cannula through an intercostal space, and leaving the cannula there until the opening in the lung is closed. This must not be done without antiseptic precautions, and a trustworthy antiseptic dressing should be applied over the side of the chest. When it is found that air no longer tends to accumulate in the chest, the cannula may be removed, and the pneumothorax will be disposed of as in the cases belonging to the last category. Though these have been described as cases of pure pneumothorax, it must not be forgotten that there is always a certain amount of blood thrown out into the pleura, and that a considerable amount of dulness may be caused by a very small amount of blood—much more, indeed, than would be the result of the presence of an equal quantity of free fluid.

2. **Pneumothorax from rupture of a small tubercular cavity.**—Just as in the traumatic cases a small amount of blood is always present, so in those which result from the rupture of a minute tubercular cavity or emphysematous vesicle, it is not improbable that there will be a small amount of serous fluid present, implying a certain amount of acute pleurisy; still, it is the presence of air which is the striking feature of the case. It is seldom that surgical interference is required; but, if dyspnœa should be caused by the increasing accumulation of air, the treatment must follow the same lines as those just described.

The subject of tubercular pneumothorax will be returned to under heading 4.

3. **Pure hydropneumothorax**—that is to say, a condition where the fluid is perfectly clear—for I do not include under this heading those cases of hydropneumothorax where the fluid, though not strictly purulent, is opaque—must be an extremely rare affection. Almost the only genuine case of hydropneumothorax which I have seen was the result of the rupture of a hydatid of the lung. Repeated aspirations of the fluid were followed by an immediate increase of the pneumothorax, and subsequent reaccumulation of the fluid, so that it became ultimately necessary to make a free opening into the thorax, which led to a correct diagnosis being made and the removal of the cyst. I should adopt this line of treatment if a similar case were to present itself, always supposing that the presence of tubercle could be excluded; but experience of this matter is too slight to justify the laying down any very definite rules of treatment.

4. **Pyopneumothorax.**—We now come to a far more difficult class of cases. It includes those that are associated with tubercle

of the lungs, and those which are due to the rupture of an abscess of the lung arising from some other cause, or to the perforation of some ulcerating tumour of the trachea, or of the larger bronchi, or in some cases to an abscess in distant parts, such, for example, as a pericæcal abscess.

The tubercular cases are usually marked by great chronicity, the relative proportions of air and fluid vary to any extent, and the character of the fluid from that of slightly turbid serum to that of moderately thick pus. Sometimes the amount of air is very large, and the amount of fluid, which is very insignificant, shows no tendency to increase. Patients with this condition of things often exhibit scarcely any symptoms save that of shortness of breath, and are able to follow a quiet occupation for months or even years. These cases are better left alone. The extraction of the air or the fluid, or both, is followed by very temporary relief, and a free incision is almost certain to be followed by disastrous results. This conclusion is arrived at as the result of sufficient personal experience. After an incision the patient is for a time improved, but the lung shows no tendency to expand, and before long, in spite of great precautions, it usually happens that septic changes occur. It has seemed as if the tubercular processes were stimulated to increased action as the result of the operation, and before very long the patients have fallen victims to the combined effects of tuberculosis and sepsis.

If, however, the amount of the fluid is large and increasing, something must be done to relieve the dyspnœa. Aspiration should be first tried, and it should be repeated from time to time as the fluid accumulates; but if no good result from this, the following method of treatment may be adopted. The patient should be placed on his back close to the edge of the bed, or rather with his side projecting slightly beyond it. Two perforated needles are inserted into the chest, one into the anterior part, after ascertaining that the lung is not adherent at the spot selected for its introduction, and the other at the posterior part of the chest—say at the eighth or ninth interspace, below the angle of the scapula. To each needle is attached an india-rubber tube. That in connection with the anterior one passes to the bottom of a large bottle containing warm boric acid lotion, which has been prepared with sterilised water, temperature 100° F., and to the other needle is attached a tube which is either connected with the aspirator or is allowed to pass into a basin which is placed upon the floor. The fluid is thus allowed to drain from the chest, and its place is taken by the boric acid lotion. When this comes through quite clear, the anterior tube may be removed, and then as much as possible of the boric acid lotion is to be evacuated, either by the aspirator or the syphon action of the posterior tube, as the case may be. I have tried in similar cases the injection of tincture of iodine into the chest, but I cannot say that it has given very satisfactory results.

In those cases of pulmonary tuberculosis in which the accumulation of air causes trouble to the patient, it may sometimes be necessary to remove it by aspiration. This occasionally benefits the patient for some considerable time, so that it is not only desirable to perform it but to repeat the process occasionally. Sometimes the improvement only lasts for a few hours, and the dyspnœa again becomes very urgent. Under these circumstances relief may be gained by inserting a minute cannula through an intercostal space and applying an antiseptic dressing over it; but it is most likely that in these cases a free incision will ultimately be necessary. Such a case occurred at the Brompton Hospital, in which at the post-mortem there was found, at the posterior part of the lower lobe, a small tubercular cavity the wall of which was projecting through an opening into the pleura. In this instance the patient died within a few days after the rupture.

5. **Pyopneumothorax resulting from decomposition of pus, &c.**—With regard to the cases of pyopneumothorax resulting from the rupture of other abscesses into the pleura, and those included under the fifth category, where the gas is simply the result of decomposition, nothing need be said, except that we must be guided by the rules that have been laid down for the treatment of empyema, the presence of the pneumothorax being only an accidental and, so to speak, immaterial complication. It should, however, be stated that some physicians doubt the possibility of the occurrence of this variety of pyopneumothorax: that is, they assert that pyopneumothorax does not occur unless there is a communication between the pleura and the lung or some other air-containing viscus such as the stomach or intestine. It is a point, I imagine, very difficult to settle. Personally I have no doubt that gas may be met with in the cavity of the peritoneum without a rupture of the intestine, and I am strongly of opinion that it may also be found in the pleura.

R. J. G.

CHAPTER LVII

HYDROTHORAX

(DROPSY OF THE PLEURA)

PAGE

Etiology 645

Character of the effusion 645

Morbid anatomy 646

Symptoms 646

Physical signs 646

Diagnosis 647

Course 647

Treatment 647

Etiology.—An accumulation of serous fluid in the pleural cavity, apart from inflammatory changes, may occur under a variety of conditions, of which chronic valvular disease of the heart and Bright's disease are the most common. Cardiac failure, from whatever cause arising, may be accompanied by dropsy, and in this the pleural cavities may share. Hydrothorax may also be present as a complication of mediastinal tumour or aneurysm, and often accompanies the later stages of cirrhosis of the liver. The distribution of the fluid in conditions which ought apparently to affect all the serous cavities of the body to an equal degree is extremely variable, and for this no obvious explanation can be offered. In renal disease it is commonly bilateral, and in cardiac failure it should theoretically be so, but in the latter case it is not infrequently limited to one side. An intra-thoracic tumour, originating possibly in the bronchial glands, may give rise to either unilateral or bilateral hydrothorax, from pressure upon the azygos veins.

In cases of general dropsy, in which unilateral pleural adhesions are present, the corresponding lung may be œdematous and a considerable quantity of fluid may be found in the opposite pleura.

Character of the effusion. The effusion consists of a clear serous fluid in which a few flakes of fibrin may be present; these may, as suggested by Hamilton,[1] be formed after death.

The amount of albumen found in dropsical effusions varies not only in different individuals but in the different parts of the body, but there is a certain fairly constant relation between the quantity found in fluids taken from the various serous cavities, as all analyses show that the proteids are in excess in pleural effusions. This is attributed by James[1] to the fact that the negative pressure of the pleural cavity tends *cæteris paribus* to aspirate a liquid rich in albumen.

The following analyses of the quantity of albumen may be quoted:

	Pleura	Peritoneum	Cereb. vent.	Subcutaneous
Schmidt	28·5	11·3	8	3·6
Hoppe-Seyler	42·41	32·32	—	17·83
Lehmann	18·52	10·44	5·64	—

The amount of the effusion varies from a few ounces to many pints, and the quantity present on the two sides often differs greatly.

Morbid anatomy.—If the effusion is of recent date, the pleura retains its ordinary glistening appearance, but if it is of long standing the visceral layer is almost invariably slightly thickened and of a dull greyish-white tint. This change is due to an exudation from the surface, which becomes organised and forms a whitish fibrous film, similar to that found in the peritoneum in ascites of prolonged duration. The exudation can be peeled off, and when held to the light is seen to be of unequal thickness in different parts. The prolonged contact of fluid with a serous membrane appears to excite a slight degree of inflammatory change which leads to the appearance just described.

The lung, either as a whole or more often the lower lobe only, will be found collapsed, and where the effusion is of long standing and the pleural covering is thickened, considerable pressure from within is required to produce complete expansion of the organ. The anterior margin of the lower lobe in such a case is often prolonged into a finger-like process.

Symptoms.—When in a case of general dropsy from chronic valvular disease the dyspnœa is noticed to be increasing, but little thought is as a rule given to the presence of hydrothorax as the possible cause of the change. This is more often attributed to the progress of the primary disease, and the condition of the pleural cavities is overlooked because 'the patient is too ill to be examined.'

Pathological experience led the writer many years ago to the conclusion that, next to an unsuspected tuberculosis, an enormous hydrothorax is the most common revelation of the post-mortem room. Since then, undeterred by the presence of orthopnœa, a distressing sense of oppression, cyanosis, or even signs of impending death, he has made it a rule to carefully examine the chest in all cases. The result has been the frequent discovery of hydrothorax, and great relief or even temporary recovery has not uncommonly followed the withdrawal of the fluid by paracentesis.

The **physical signs** are those of fluid in the pleura, but the lesser density of the effusion and the absence of the thick layer of exudation usually present in serofibrinous pleurisy render the signs less obvious, and account in part for the fact that the presence of hydrothorax is so often overlooked.

---

[1] *Medical Times and Gazette*, 1880: 'Transudations and Exudations.'

There is also less distension of the side and the intercostal depressions are rarely obliterated. Vocal fremitus is absent, and on light percussion a dull note is obtained; faint tubular breathing may be audible over the site of the effusion, and the whispered voice (Bacelli's sign) may be well conducted. Friction sound is absent.

If the effusion is bilateral and equal in amount on the two sides—a condition which is, however, rarely met with—the position of the heart will not be affected.

Diagnosis.—The clinical characters which differentiate hydrothorax from pleurisy with effusion are briefly the history of the case, the absence of pain and fever, and the presence of some condition sufficient for the production of dropsy. Too much stress should not be laid upon the fact that the effusion is (or rather should be) double in hydrothorax.

Course.—In a case of chronic valvular disease with recent cardiac failure, the serous fluid may accumulate rapidly, but in renal dropsy a moderate amount of effusion is often present in both pleuræ for a long period preceding the death of the patient.

Treatment.—As will appear from what has been stated, the writer is strongly of opinion that in cases of cardiac failure from valvular disease, more attention should be given to this condition. The failure of the heart may be due to some temporary cause; if so, the removal of the fluid by paracentesis is followed by great relief to the dyspnœa and by recovery of such health as is possible in a case of the kind. Should the fluid reaccumulate the operation may be repeated. When summoned to a patient with chronic mitral disease, who is in bed with enormously swollen legs and general dropsy of the serous cavities, a surprising change may follow if he is removed to an arm-chair, the legs drained with Southey's tubes, the fluid withdrawn from the pleura by paracentesis, and the right side of the heart relieved by venesection. After these measures have been adopted, digitalis and other remedies, which previously appeared to have but little effect, may be observed to regain their power for good.

Absolute cleanliness of the skin and the use of a needle which has been boiled for fifteen minutes in a steriliser are precautions as necessary in paracentesis for hydrothorax as in that for the removal of an inflammatory effusion.

The general treatment of cases of dropsy must be conducted on principles which are well known and do not need description.

J. K. F.

CHAPTER LVIII

HÆMOTHORAX

PAGE
Etiology 648
Symptoms 649
Physical signs 649
Course 649
Diagnosis 649
Prognosis 649
Treatment 649

Etiology.—A collection of blood in the pleural cavity may occur from injuries to the chest wall with fracture of the ribs and laceration of the lung. Hæmothorax, due to these and other traumatic conditions, is dealt with in the chapter on Injuries of the Pleura and Lung (*vide* p. 538).

The rupture of an intra-thoracic aneurysm may lead to the sudden escape of an enormous quantity of blood into the pleural cavity. If an aneurysm perforates a mucous membrane such as that of the trachea or œsophagus, the fatal event may be preceded for some days by small bleedings, but rupture into a serous cavity is generally attended by sudden death. This statement, however, applies more decidedly to the pleura and peritoneum than to the pericardium.

Rupture of an intra-thoracic vein is also a rare cause of hæmothorax.

In the chapter on Acute Pleurisy (*vide* p. 555) the subject of hæmorrhagic effusions, which may be either passive or the result of inflammation, is considered, and their frequent association with cancer and tubercle of the pleura is referred to. It is not, however, usual to speak of a serous or an inflammatory effusion which is blood-stained as a hæmothorax. In a case recently under the care of the writer fatal hæmorrhage into the pleural cavity occurred after an operation for pyopneumothorax owing to the rupture of a small aneurysm of the internal mammary artery. Five other unruptured aneurysms were found on the same vessel; they closely resembled the aneurysms of the pulmonary artery, so commonly met with in cases of tuberculosis of the lungs, and were produced by the extension to the walls of the vessel of the inflammatory process which involved almost the whole of the pleura.

A pulmonary infarction has been known to rupture and allow the escape of blood into the pleura.

In certain diathetic diseases, such as scurvy and purpura, hæmorrhage may take place into the serous cavities.

The **symptoms** are common to those of hæmorrhage generally, viz.: syncope, pallor, coldness of extremities, &c., and their severity depends upon the amount of blood extravasated and the rapidity with which it escapes from the vessels.

In addition to the rapid breathing which ordinarily attends a considerable loss of blood, there is laboured dyspnœa due to the compression of the lung. Pain may be absent.

The **physical signs** are those of pleural effusion, primarily at the base and extending upwards as the quantity of blood increases. Friction is not heard. The note obtained on percussion over blood which has coagulated is absolutely dull, and as the film thus formed may be very thin, a very small effusion of blood may give rise to physical signs which would only be produced by a very large serous effusion. If both air and blood are present—a very rare condition—and the blood remains uncoagulated, it is possible that the upper level of the dulness may alter with the position of the patient, but usually the blood coagulates.

Course.—In traumatic cases the blood, as a rule, quickly coagulates. The serum is then slowly absorbed, and at a later period the clot also. If septic infection of the pleura occurs, acute inflammation follows, and an empyema results.

Diagnosis.—The presence of an effusion will be recognised by the ordinary signs, fever will be absent, and there may be symptoms of internal hæmorrhage. When a pleural effusion takes place very quickly after the receipt of an injury to the chest, the inference that hæmorrhage has occurred is a fair one.

Prognosis.—This depends to some extent upon the cause of the hæmorrhage. If a patient is known to have a thoracic aneurysm, and rupture takes place into the pleura, death will probably occur before the necessity arises for giving a prognosis.

After the chest has been opened for empyema or pyopneumothorax, if hæmorrhage occurs, the blood may come from a wound of an intercostal artery, and in that case the bleeding will be easily controlled; but the possibility of its arising from the rupture of an aneurysm of the internal mammary artery must be borne in mind. The latter condition is almost certain to lead eventually to a fatal termination.

Treatment.—An expectant attitude is usually most advisable, and great caution is necessary before deciding on operative interference when the source of the bleeding is unknown.

If the dyspnœa becomes very urgent, and life is threatened, it may be necessary to draw off as much of the effusion as suffices to give relief; but it is rarely wise to go beyond this except in traumatic cases.

CHAPTER LIX

CHYLOTHORAX

(CHYLOUS PLEURISY)

PAGE
Pathology 650
Clinical case 651
Composition of chylous fluid 653
Symptoms 654
Physical signs 654
Course and prognosis 654
Diagnosis 654
Treatment 655

Cases in which an effusion of a chylous character is present in the pleural cavity are usually described under the heading 'Chylous Pleurisy,' but as the effusion is passive and non-inflammatory the term 'pleurisy' is not strictly appropriate. 'Chylous hydrothorax' has also been employed, but is open to more decided objection. The term here used appears to be preferable, as it indicates the nature of the effusion and also implies the absence of inflammation. The effusion may be limited to the pleura, or a similar fluid may also be present in the peritoneal cavity.

Pathology.—In the majority of cases chylothorax is caused by occlusion of the thoracic duct, but it may arise from rupture of the thoracic duct or of the receptaculum chyli. A case is recorded in which chylothorax resulted from tubercular disease of the thoracic duct. It is not always possible to demonstrate post-mortem the site of the lesion, or even to find the duct or the receptaculum chyli. In a considerable number of recorded cases the obstruction has occurred at or about the entrance of the duct into the left subclavian vein. The nature of the obstruction present in a case reported by Dr. Cayley[1] is described thus: 'At the junction of the thoracic duct with the left subclavian vein the former suddenly became much narrowed, and its coats thickened, and just at its mouth a fibrinous granular vegetation, which almost completely obstructed the opening of the duct, was attached to the lining membrane of the vein. Below this there was a firm, yellow, cylindrical coagulum.'

In a case also under the care of Dr. Cayley, and reported by Dr. Sidney Martin,[1] the obstruction was secondary to thrombosis and contraction of the veins at the root of the neck on the left side.

In this case the abdomen contained 180 oz. of chylous fluid ; the lymphatics of the intestine, especially those of the small bowel, were dilated and contained milky fluid. Neither the thoracic duct nor the receptaculum chyli could be found. The lymphatics of the left pleura, and to a less extent those of the right also, presented a milky arborescent appearance. The left pleura contained 10 oz. of milky fluid, on the right side the surfaces were found adherent, especially over the lower thirds. The lungs showed general increase of the connective tissue and distended lymphatics.

In a case reported by Dr. Turney[2] the primary lesion was a cancerous growth of the pylorus, 'composed of large epithelial cells of a spheroidal shape, mostly filled with colloid material.' There were secondary deposits of the same nature in nearly all the superficial glands of the body. The thoracic duct was greatly dilated in its whole course. Near its termination the duct divided into two branches, which led to the junction of the left subclavian and internal jugular veins. The orifices of the duct in the vein were surrounded and blocked by dry, partially decolorised adherent clot, which almost immediately on either side tailed off into ordinary soft black coagulum. The thrombus was adherent to the wall of the vein, but could be fairly easily separated. The intima was unaffected. Neither the subclavian nor the internal jugular vein, though surrounded by large glands, was compressed by or adherent to them. The obstruction was believed to be due to the impaction of a fragment of the new growth in the orifice of the duct. The lymphatics of the intestine, especially those of the ileum, and also those of the pericardium and right pectoral muscles, were distended with chylous fluid. The right pleura contained two pints of milky fluid ; the membrane was white and opaque. The left contained a pint of turbid serum with fat in suspension, but the fluid was not milky. In the peritoneum there were four ounces of milky fluid, but no signs of peritonitis. There was collapse of portions of both lower lobes, and at the right apex there were two calcareous nodules with scars in the overlying pleura (? arrested tuberculosis).

Clinical case.—The following is an abstract of the notes of a case recently under the care of the writer (J. K. F.)

Fanny J., æt. 31, machinist, admitted to the Brompton Hospital on September 30, 1895. No family history of tuberculosis. Winter cough for ten years. In December 1894 the patient suffered from bronchial catarrh, which was followed by shortness of breath. This has continued, but has been worse since June 1895. The most prominent symptoms have been cough with expectoration, emaciation, pain on the right side of the chest, and night sweats. There has been no hæmoptysis.

---

[1] *Path. Soc. Trans.* vol. xlii. p. 93.

[2] *Ibid.* vol. xliv. p. 1.

On admission—Pulse 92, resp. 20, temp. normal. Right side—Dulness and absence of breath sounds and of vocal fremitus, from just above the angle of the scapula posteriorly and from the nipple anteriorly, to the base; feeble breathing above the level of the dulness. Left side—Expansion increased; respiratory sounds harsh at apex, elsewhere signs normal. Cardiac impulse in fifth interspace in left mammary line.

Oct. 5.—An aspirator needle was inserted in the seventh right interspace, near the angle of the scapula, and passed in several directions, but no fluid was withdrawn.

On Nov. 5 crackling râles were first noted above and below the left clavicle.

On Nov. 20 the same aspirator needle was inserted in the seventh right interspace and milky fluid withdrawn (*1st Aspiration*).

Nov. 25.—*2nd Aspiration.* 65 oz. of milky fluid withdrawn. Râles at left apex extended to third interspace.

Nov. 30.—Pleuritic friction sound over right upper lobe, front and back.

Dec. 12.—*3rd Aspiration.* 74 oz. milky fluid withdrawn.

Dec. 23.—Dulness at left apex to second rib. The râles were now audible in the upper part of the left axilla.

1896, *Jan.* 2.—Pain in chest returned as fluid re-accumulated. Has lost 5 lbs. in weight since admission.

Jan. 16.—*4th Aspiration.* 45 oz. milky fluid.

Jan. 30.—Dyspnœa urgent. Marked inspiratory recession of epigastrium, lower interspaces, and infraclavicular regions; slight hæmoptysis.

Jan. 31.—*5th Aspiration.* 80 oz. of fluid presenting a pinkish tint from the presence of blood withdrawn.

Feb. 8.—Patient discharged at her own request. Death occurred shortly after arrival home.

The temperature was normal or sub-normal throughout the illness. The number of respirations varied between 20 and 34 per minute, but on the day preceding the patient's discharge and at the time of leaving they were 40 per minute. The total quantity of fluid withdrawn was 264 oz. The sputum was frequently examined for tubercle bacilli, but none were found.

The post-mortem examination was made by the writer with the assistance of Dr. J. J. Perkins, under the most unfavourable circumstances possible. Leave was only obtained to examine the thoracic viscera, the incision allowed being only such as sufficed for this. The light was very bad. The abdominal organs were felt, and to a slight extent seen, through the diaphragm after the latter had been cut away. Nothing abnormal could be detected in the abdomen. It is impossible to state whether chylous ascites was present, but, as far as could be made out, there was no fluid in the peritoneal cavity. The diaphragm was cut away widely, in order not to injure the central structures, and the thoracic duct was divided as low down as possible, but its lower part and the receptaculum chyli were left *in situ* and were not even seen. The contents of the thorax were removed *en masse.* It was unfortunate that the receptaculum chyli and the whole of the duct could not be removed, and also that the structures on the abdominal side of the diaphragm could not be carefully examined.

The right pleura contained several pints of chylous effusion, but there were no means of measuring the exact quantity. No opening from the duct into the pleural cavity could be seen. The left pleural cavity, the pericardium, and the heart were normal. Miliary tubercles of a very fibroid type studded the apex of each lung, especially the left, and formed a continuous mass in the extreme upper part of each organ. Elsewhere the lungs were unaffected. There was no dilatation of the lymphatics of the pleura.

The œsophagus, the aorta, and all the mediastinal contents were normal, and there was no enlargement of the mediastinal glands. Both the thoracic ducts were dissected out completely, and their openings into the great veins clearly seen and proved to be patent by the injection of coloured water. The ducts were not dilated and their walls were apparently normal throughout. No stricture was found. No rupture could be seen in the wall of the main duct, but the connective tissue of the lower part of the posterior mediastinum around the ducts was looser and more areolar than usual, *i.e.* the meshes of the connective tissue were very much wider than normal, and when water was run into the tissue it floated out in the form of ragged festoons. Corresponding to this spot the surface of the parietal pleura on the right side had a curious worm-eaten appearance, and it was observed that when water was played on the opposite surface the fluid percolated through in drops, and ran down into the pleural cavity.

It appeared probable that a minute perforation of the thoracic duct existed, and that the chyle had infiltrated the mediastinal tissues, and through these worm-eaten spots in the pleura had made its way drop by drop into the cavity.

Composition of the fluid in chylothorax.—In chylous fluid a large quantity of fat is present in the form of an emulsion. Microscopically, numerous minute refractive granules are seen, and a few leucocytes may be found. Chemically it contains coagulable albumen and globulin, fat and inorganic salts.

In Dr. Sidney Martin's paper a table will be found giving the percentage analysis of various chylous extravasations compared with that of chyle. The following is the analysis (made by Dr.

—	Sp. gr.	Water	Total solids	Proteid	Fat	Salts	Extractives	Sugar	Error of analysis
Chyle .	—	90·5	9·5	7·1	0·9	0·48	1·0	—	—
Dr. Martin's case	1·022	92·98	7·02	4·46	1·78	—	—	·00	0·3
Dr. Fowler's case	1·023	91·66	8·33	5·26	2·12	0·68	—	0·20	0·076

Martin) of the fluid in the case recorded above, and of the case which he has described.

Symptoms.—The occurrence of the affection is in most cases more or less of the nature of an accident, and the symptoms are necessarily to a great extent those of the primary lesion; we shall therefore only consider those which can be directly attributed to the peculiar character of the effusion.

Emaciation, which might naturally be expected to follow such a considerable loss of chyle, does not appear to occur from that cause, as it is not met with except in cases in which a loss of weight might be expected from the nature of the original disease, *e.g.* in cases of cancer. In Dr. Martin's case the body is described at the autopsy as being 'well made and well nourished.' In the writer's case the patient weighed 6 st. 12 lb. on October 8, and 6 st. 7 lb. on January 16, a loss of 5 lb. only. During that period 264 oz. of fluid of a sp. gr. 1·028, which would weigh more than 15 lb., were withdrawn from the body by paracentesis.

The symptoms are not apparently influenced by the peculiar character of the effusion in the pleura, but in the case narrated the degree of dyspnœa at an early period seemed somewhat out of proportion to the quantity of fluid. The pain was invariably increased as the quantity of fluid became considerable; this was probably due to increased tension within the pleural cavity, as the pain was always immediately relieved by paracentesis. It is interesting to note that the development of a tuberculosis of the fibroid type did not cause pyrexia, the temperature remaining normal or sub-normal almost throughout the illness (it once touched 100° F.)

The **physical signs** are those of pleural effusion. In the case described, the dulness was almost always somewhat higher in the axilla than in the nipple line, but at one examination the following note was made: 'The upper border of the dull area is nearly horizontal, but slightly higher behind than in the nipple line.'

The circumference of the affected side was invariably increased below the nipple, but on some occasions it was slightly less above that level than on the opposite side. The symptoms and physical signs here given are those observed in a case of doubtful origin and cannot be regarded as typical of the condition.

Course and prognosis.—The prognosis is in all cases very unfavourable, but the course of the illness is necessarily influenced to some extent by the nature of the primary lesion. We are not indeed aware that recovery has ever occurred, but if the obstruction is slowly produced, it is quite possible that, as suggested by Dr. Cayley, an anastomosis may be established through the right lymphatic duct.

The duration of the illness in Dr. Martin's case was six months, in the writer's eight and a half months.

Diagnosis.—It is extremely improbable that the nature of the effusion will in any case be diagnosed before paracentesis has been performed, but by a process of exclusion it might be suspected to be chylous if with a normal temperature and the absence of a history of acute pleurisy, and of any condition likely to give rise to hydrothorax, fluid was found to be present in the pleural cavity.

Treatment.—The fluid should be removed as often as it accumulates in sufficient quantity to cause suffering, but it is inadvisable to do more than this.

J. K. F.

CHAPTER LX

TUMOURS OF THE PLEURA

PAGE
Primary growths 656
  1. Endothelioma or endothelial cancer 656
  2. Sarcoma 657
  3. Fibroma 657
Secondary growths 658
Clinical cases 658
  Cancer 658
  Sarcoma 660
  Fibroma 661
Symptoms 662
Physical signs 662
Course and duration 663
Diagnosis 664
Treatment 664

New growths rarely originate in the pleura, but secondary infiltration of the serous membrane is not uncommon in cases of malignant disease.

Primary tumours.—The following varieties of new growths are met with:

1. Endothelioma, or endothelial cancer.
2. Sarcoma.
3. Fibroma.

Pathology and morbid anatomy.—The exact position among neoplasms of the growth to which the term '*endothelioma*' is applied by some writers, and 'endothelial cancer' by others, is uncertain. Histologically, such a growth cannot be separated from cancer. When occurring in the pleura, it has been thought that it may arise from the multiplication of the endothelial cells of the lymphatics, but this is considered by Ziegler to be doubtful. As the surface epithelium of the serous membrane is in a state of active proliferation (Neelsen), and gives rise at least to some of the columnar elements of the growth, it is more natural to refer the development of the neoplasms generally to the proliferation of the epithelium of the primitive body-cavity, and to regard such growths as genuine examples of carcinoma (Ziegler: MacAlister's Trans., Chapter xxxii. sec. 358).

The same view is taken of the nature and site of origin of the growth in a case of primary tumour of the pleura recorded by Dr. Coats ('Glasgow Med. Journal,' July 1889, p. 15).

These growths often form multiple white flattened nodules, connected by bands of similar structure, and on microscopical examination show nests and clusters of epithelial cells lying in a dense fibrous stroma, containing round and spindle-shaped nuclei. Alveoli, lined by epithelioid or cylindrical or cuboidal epithelial cells, may be present throughout the growth, or chiefly at the periphery; or the cells may be arranged in columns. The appearance in some cases of a growth of the type of a cylindrical epithelioma has suggested that the real site of origin may be the mucous glands of the bronchi, but this view has been negatived by the absence of invasion of the tubes.

In the case above referred to, reported by Dr. Coats, there were numerous tumours, which by coalescence had formed a layer of new tissue of considerable thickness. The sac of the pleura contained 140 oz. of blood-coloured fluid, which had deposited a loose brown coagulum. No growths were present elsewhere. Microscopical examination showed the growth to be a superficial cancer with a stroma enclosing epithelial cells, many of which were fatty. In this case the growth was not deep in the substance of the pleura, and did not involve the muscles or ribs, but both these structures may be implicated. A cancerous growth may invade the tissue of the mediastinum and involve the bronchial glands, and extend into the lungs along the peribronchial tissues.

Sarcoma.—Nearly all the varieties of sarcoma may be met with in the pleura, but probably the round-celled growth is most common. A sarcoma may appear as a thick, dense, fibrous layer, lining the whole pleural cavity, more marked perhaps on the parietal than on the visceral layer; sometimes, as in a case of this kind examined by the writer, enormous rounded tuber-like masses of new growth may project into the cavity, and a dense infiltration of the membrane by a soft whitish growth may also be present.

As will be seen from the cases cited subsequently, a blood-stained pleural effusion and acute inflammation of the serous membrane are changes commonly present in these cases.

Secondary infiltration may be present in the mediastinum, the bronchial, tracheal, œsophageal, retro-peritoneal and supra-clavicular glands, and, either by extension or metastasis, the growth may involve the opposite pleura, the pericardium, heart, peritoneum, liver, spleen, kidneys, and intestines.

Fibroma.—The true nature of the cases thus described must be considered doubtful. Some may possibly be examples of pure fibroma; it is, however, more probable that they are sarcomata, in which the fibrous elements predominate to an unusual degree. Dr. Steven (' Mediastinal Tumours,' p. 67) has recorded a case which he regards as one of fibroma of the mediastinum, in which the association with rheumatism was a marked feature. A case which appeared to be one of fibroma of the pleura will be described later.

Secondary tumours.—Malignant growths in the lung or mediastinum often infect the pleura, and secondary infiltration of the membrane is especially common in cases of cancer of the breast, but may occur when the primary growth is variously situated in other parts of the body.

CLINICAL CASES

Cases of primary tumour of the pleura are so rarely met with that individual experience of them is necessarily limited. We shall, therefore, before describing the symptoms and physical signs which may be present, give abstracts of the clinical records of some typical examples of the affection.

Cancer.—Henry T., æt. 36, a bootmaker, was admitted into the Middlesex Hospital on July 30, 1890, under Dr. W. Pasteur.

Family history.—Father died, æt. 38, from an 'internal tumour.'

Previous history.—Health has been good, with the exception of an attack of bronchitis ten years ago, for which he was an in-patient in this hospital.

The *present illness* commenced three months ago with a slight cough, which has continued. He has had *no pain* in the side, but has suffered from breathlessness on exertion for the last six weeks.

On admission.—Well nourished. Temperature normal. Pulse 88, respiration 26. The *right side* of the chest appeared slightly bulged, and did not expand on inspiration. Vocal fremitus was absent, except over a small area beneath the clavicle, and over the apex posteriorly, and in the interscapular region. There was complete dulness, and an increased sense of resistance over the right side, up to the second rib in front, and to the spine of the scapula behind, and over the same area the breath sounds were absent. Above this level they were feeble, but over the root of the lung they were of bronchial quality. The vocal resonance about the angle of the scapula was high pitched and nasal in character (ægophony). The cardiac impulse could neither be seen nor felt, but was localised by auscultation in the fifth interspace, one inch external to the left mammary line. No murmur was audible. The lower edge of the liver was felt nearly one inch below the costal margin.

On July 31, thirty-five ounces of blood-stained fluid were withdrawn by paracentesis from the right pleural cavity. Sp. gr. 1·014—highly albuminous.

After the operation, vocal fremitus was present over the right side, except at the extreme base; the cardiac apex was felt one inch within the nipple line; faint breath sounds became audible, and the ægophony disappeared.

On August 2 the temperature was 103° F.; respirations 54, pulse 120; the face was dusky, the lips and ears cyanosed. A dry friction sound was audible at the angle of the right scapula.

Paroxysmal cough, with abundant watery, frothy expectoration followed.

August 9.—Sputum examined for tubercle bacilli, but none found.

August 11.—The patient was seen by the writer (J. K. F.), who agreed that 'the balance of evidence was in favour of a new growth as against tubercular pleurisy.'

August 18.—After consultation, paracentesis was performed, and 35 oz. of almost pure blood were removed. The patient shortly afterwards had a severe attack of dyspnœa, from which he recovered, but died after a subsequent attack on the following day (August 19).

Autopsy.—Body well nourished and well developed. The right pleural cavity contained 80 oz. of fluid, a considerable part of which was blood. This had subsided to the base of the cavity. The visceral layer of the right pleura was completely studded with small eminences, resembling beads of white wax. Over the lower third of the parietal layer, and particularly over the diaphragm, there were similar growths. The growth did not extend beneath the pleura, except at one or two points, where it dipped slightly into the right lung. None of the neighbouring organs or structures were invaded, and no secondary growths were present. The right lung was small and carnified; the left was congested and œdematous. A milky fluid exuded on pressure from some enlarged and pigmented glands at the root of the left lung.

In this case the pleural origin of the growth could not be doubted, as the degree of infiltration of the lung was trifling. It is unfortunate that there was an omission to note in the post-mortem report the results of the microscopical examination of the portions of the growth which it is stated were reserved with that object. The case is headed 'Cancer of the Pleura.'

In some cases in which both the lung and the pleura are involved it may be difficult to determine the site of origin of the growth. The following is an example of this kind. The growth was believed to be a primary cancer of the pleura, with secondary infiltration of the lung.

Case II.—Thomas M., æt. 44, was admitted into the Middlesex Hospital on March 16, 1894, under the care of Dr. Fowler, and died on April 23. There was no history of tubercular disease in the family. The patient had hitherto enjoyed good health, and could not assign any cause for his present illness, the onset of which dated from Christmas 1893, when he began to suffer from cough and shortness of breath. These symptoms had lately increased in severity. The expectoration had been white and frothy; there had been no hæmoptysis. There had recently been emaciation and loss of appetite. On admission he was fairly well nourished. The chest was of the emphysematous type, with a wide epigrastric angle. There was pulsation in the epigastrium and in the veins of the neck, which were much dilated. The cardiac impulse could not be localised, but the sounds were most distinct at the normal position of the apex beat. The movement of the right side was deficient below the fourth cartilage. On percussion, there was hyper-resonance over the right apex in front and behind, but there was dulness from the lower border of the third rib in the mammary line, a rather higher point in the axilla, and the angle of the scapula posteriorly, downwards to the base. Over the dull area the breath sounds were feeble, with prolonged faintly blowing expiratory sounds in front. The vocal fremitus was diminished. There was a pleural friction sound in the left axilla. The edge of the liver could be felt below the costal margin. The urine was free from albumen and sugar. Pulse 96; resp. 35; temp. 97°.

Note on March 31.—Inspiratory recession of interspaces in both axillæ; no expansion on right side. In front, absolute dulness from first rib to base, but fremitus distinct; breath sounds are audible all over dull area, but they become gradually fainter towards the base; no adventitious sounds are audible. Posteriorly vocal fremitus absent at base, dulness from middle of inter-scapular region downward; voice sounds ægophonic. Feeble breathing back and front on left side, with crepitation.

April 6.—The patient is cyanosed, and there is more marked distension of the veins of the right side of the neck.

April 17.—Forty ounces of blood-stained fluid were withdrawn from the right pleura.

April 23.—Dyspnœa more urgent. Death.

P.M.—Body emaciated, œdema of lower extremities. Trachea and bronchi filled with blood-stained, frothy mucus. Right lung considerably indurated, numerous white masses of new growth scattered through upper and lower lobes. A small cavity situated in one of these white masses leads to a deep scar on the surface. Lung tissue firm and tough; bronchi of lower lobe dilated. Similar white mass in anterior and lower part of left upper lobe. Sixty-two ounces of blood-stained serum in right pleura, 32 oz. in left; pericardial sac contains 40 oz. of fluid.

Microscopical examination of the lungs shows the presence of an epithelioma with squamous cells, some with very large single nuclei, others containing many nuclei. The pleura is thickened and fibrous, and in it are irregular columns of epithelial cells, suggesting an invasion of the lymphatic spaces. The lung tissue is extensively invaded with epithelial new growth and shows inflammatory thickening. No evidence of tubercle.

Sarcoma.—The following case is reported by Dr. de Havilland Hall.[1]

H. A., æt. 19, a clerk, admitted to the Westminster Hospital December 5, 1879.

Previous history.—Health good up to nine weeks ago, when he caught cold whilst playing cricket. Onset with languor and loss of flesh (about one stone); shortness of breath on exertion, and slight cough without expectoration followed. No hæmoptysis. There has been slight tenderness and pain in the left side and left shoulder, but the pain has never been severe, and he has not felt very ill. Frequent vomiting since November 28.

---

[1] *Clin. Soc. Trans.*, vol. xiii. p. 200.

On admission.—Right side resonant, breath sounds puerile. Left side immovable, marked rounding and bulging of interspaces. Vocal fremitus not entirely absent. Absolute dulness, crossing median line, below second rib in front and spine of scapula behind. Resonance above these levels, 'boxy' note in front, tympanitic behind; absolute dulness below. Breath sounds faint at apex and along the spine, inaudible elsewhere; a slight creak audible at the angle of the scapula. Cardiac impulse one inch below and internal to the right nipple; no murmurs; no albumen in urine. Pulse 104; respiration 24; temp. 100° F.

December 7.—Paracentesis of left chest, needle inserted in three places, no fluid obtained; needle blocked with 'cheesy' material.

December 8.—Incision along upper border of sixth rib in mid-axillary line. Solid cheesy material found filling pleura.

December 22.—Urgent dyspnœa; collapsed. Pulse 102; respiration 39. Cardiac apex outside right nipple. Expectoration viscid, frothy, rusty. Died on following day.

Quantity of urine passed on thirteen days varied between 7 oz. and 34 oz.; daily average 20 oz. The temperature varied between 98°-99° in the evening, and 94·4°-101·2° in morning.

Autopsy.—The left side of the thorax was completely filled with new growth, situated in the cavity of the pleura, and extending two inches beyond the median line at the level of the second rib. The diaphragm was displaced downwards to the level of the eleventh rib. On section, the upper fourth of the growth was glistening, pale yellow in colour, and of gelatinous consistence; below this there was a caseous layer, then an area occupied by softened portions infiltrated with black blood clots; the lower fourth was firm and white. Secondary nodules, white and soft, were situated along the spine behind the parietal pleura. The left lung was collapsed, attached to the tumour, but not infiltrated by it. Slit-like perforation in left bronchus from the pressure of the growth. Upper and middle lobes of right lung congested, lower lobe hard, presents appearance of damson cheese on section. There were no secondary deposits in any organ. On microscopical examination the growth proved to be a round-celled sarcoma.

Fibroma.—Charlotte S. was admitted into the Brompton Hospital on March 15 in a state of collapse, and died on the following day. The history showed that six months previously the patient suffered from sudden severe pain in the epigastrium and had never been quite free from pain since. It was aggravated by food; vomiting had occurred at intervals. The urine was highly albuminous. The patient died comatose.

Autopsy.—Body thin, but apparently not much wasted. On reflecting the muscles of the thorax, the intercostal spaces on the right side, from the second to the ninth rib, were seen to be infiltrated with a firm white or pinkish new growth of very dense consistence. On removing the sternum, the lower part of the right lung, from the level of the second rib downwards, was found to be densely adherent to the ribs and cartilages from front to back; the union was so firm that it was necessary to cut the lung out of the thorax. A similar condition was present on the left side, from the level of the third rib just outside the breast to the base. After removal, the lower parts of both lungs were seen to be encased in a dense fibrous or cartilaginous capsule, which in places was two inches thick. The material was glistening and white, and somewhat resembled a section of a pine-apple. In some places there were small spaces containing clear, pale-yellow fluid. In certain areas near the diaphragm, ill-defined, opaque, yellowish-white masses were scattered through the whiter tissue. The middle and lower lobes of the right lung were partially collapsed, the interlobular septa were thickened; this change apparently extended from the pleura. The lungs elsewhere were normal. The diaphragm on the right side was converted into a dense, pinkish-white, fleshy tissue near its summit, and on the left side it was thickened. There were six secondary deposits. The large vessels of the thorax and abdomen were not compressed.

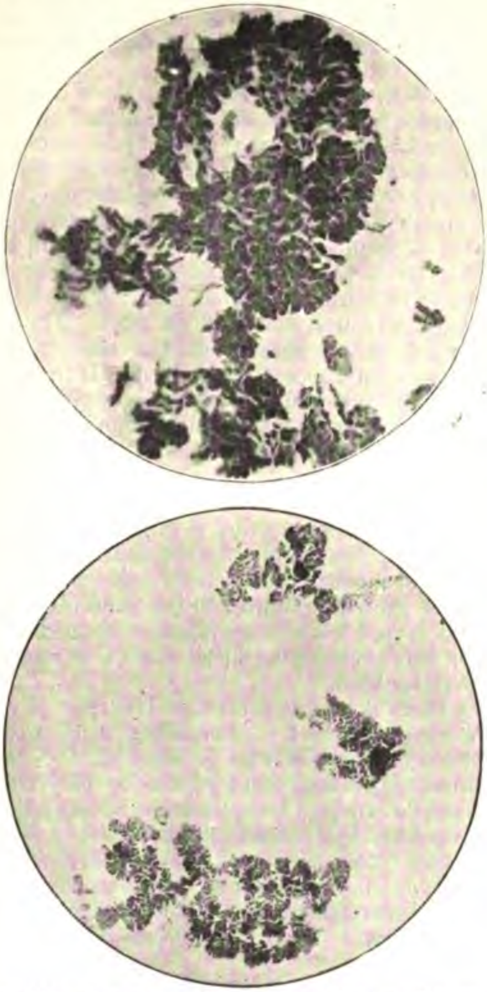
On *microscopical examination* the growth was seen to be fibrous throughout; no appearances were found which rendered it possible to regard it as other than a fibroma.

Symptoms.—The cases recorded show that the symptoms present great variety. The onset is usually insidious. Pain may be entirely absent or only slight in degree, but in some recorded cases it has been severe and persistent. In Case IV., the onset of the illness was sudden and accompanied by severe pain. Languor and loss of flesh may be the earliest symptoms, but emaciation is rarely extreme, and when death occurs the body may be described as 'well nourished.' Shortness of breath on exertion and cough are common.

In the absence of complications pyrexia is not marked; vomiting may occur; the urine may be albuminous and the quantity passed may be less than normal, and there may be clubbing of the fingers.

Physical signs.—In the majority of cases the physical signs point to a diagnosis of pleural effusion, a condition which is extremely likely to be present. The side may be rounded, bulged and motionless, with skodaic resonance above and absolute dulness below; the breath sounds being feeble or absent. In some cases, however, retraction of the affected side has been observed even when there is effusion into the pleura. The diaphragm may be pushed downwards and the heart may also be displaced, either from the presence of a solid growth or of one which has given rise to an effusion into the pleural cavity. Displacement of the heart is, however, not invariably present, even when considerable effusion has taken place. When the growth is solid, or nearly so, the vocal fremitus may not be entirely lost. Signs of pressure upon the large vessels, both venous and arterial, and upon the œsophagus and trachea are usually absent.

Course and duration.—The growth of primary tumours of the pleura is generally rapid, the duration of the disease in the cases here described being three and a half months, eleven weeks, and six months respectively.



FIGS. 147 AND 148.—GROUPS OF CELLS FROM A CASE OF TUMOUR OF THE PLEURA, FROM MICROPHOTOGRAPHS BY PROFESSOR SIDNEY MARTIN, F.R.S., SEEN UNDER A HIGH AND A LOW POWER

In a case reported by Dr. Hebb[1] the duration was about two months.

---

[1] *Path. Soc. Trans.*, vol. xliv. p. 5.

In another, reported by Dr. Pitt,[1] the symptoms had been noticed for a year before admission, and death occurred about six weeks later.

In a case recorded by Dieulafoy,[2] occurring in a man, æt. 23, the duration was apparently much longer, as violent pain had been present in the chest for some years. Paracentesis was performed twenty-five times, a hæmorrhagic fluid being withdrawn on every occasion.

Diagnosis.—As might be expected, these cases are almost invariably regarded at the outset as examples of pleurisy with effusion, and such diagnosis may be in part correct. But the absence of a history of severe pain and pyrexia, conditions which almost invariably accompany acute pleurisy, and the insidious onset of the illness, should suggest that a growth is present. Paracentesis will probably be performed, and a blood-stained fluid may be withdrawn, but after its removal the dulness may remain or the heart may not return to its normal position. Or the result of the operation may be to prove that there is no fluid. The discovery of blood-stained fluid will suggest that the case is either one of malignant disease or of tubercular pleurisy. It must, however, be remembered that in tubercular pleurisy a sero-fibrinous effusion is of more common occurrence, and that pneumococcus pleurisy may give rise to a hæmorrhagic effusion. The fluid withdrawn should be examined microscopically and by bacteriological methods, and the nature of the disease may in this manner be determined (*vide* figs. 147 and 148), but such an event is of rare occurrence. If fluid is absent the case is probably one of malignant disease of the pleura or of chronic adhesive tubercular pleurisy (*vide* p. 586).

So long as the growth is limited to the pleura the signs of pressure on the vessels and other structures so characteristic of mediastinal tumour will probably be either absent, or less marked than in cases of the latter kind.

If a portion of rib is excised (*vide* Case III.), the diagnosis will of course become clear, but such a proceeding is not advisable if the nature of the case can be otherwise determined.

Treatment.—In cases accompanied by pleural effusion but little benefit is to be expected from paracentesis, as after the fluid is withdrawn it quickly reaccumulates, and the operation may be followed by intense dyspnœa.

Urgent symptoms must be met as they arise, by the adoption of measures likely to afford relief, and in accordance with the general principles of treatment.

J. K. F.

---

[1] *Path. Soc. Trans.*, vol. xxxix. p. 56.

[2] *Soc. Méd. des Hôp. de Paris*, 1886, p. 36.

CHAPTER LXI

DISEASES OF THE DIAPHRAGM

PAGE

Etiology 665
1. Physical conditions which impede the action of the diaphragm 666
    Symptoms 666
    Physical signs 666
2. Functional disorders 666
    Spasm 666
    Symptoms 667
3. Paralysis 667
    Signs and symptoms 667
    Diphtheritic paralysis 667
    Diagnosis 668
    Prognosis 669
    Treatment 669
Diaphragmatic pleurisy 670
    Symptoms 670
    Diagnosis 670
    Prognosis 670
    Treatment 671

IMPEDED ACTION OF THE DIAPHRAGM FROM PHYSICAL CONDITIONS, FUNCTIONAL DISORDERS, AND PARALYSIS

Etiology.—The action of the diaphragm may be either impeded or in abeyance from various causes which may be thus classified:

1. Physical conditions originating within the thorax or abdomen.
2. Functional disorders giving rise to tonic or clonic spasm of the muscle.
3. Lesions of the spinal cord at or above the origin of the phrenic nerves, or changes involving the nerves only, may be followed by paralysis of the diaphragm. When one phrenic nerve only is affected the paralysis is limited to the corresponding half of the muscle.
4. Traumatic lesions causing injury, perforation, rupture, or hernia.

5. Inflammatory and degenerative changes affecting the muscular tissue.

6. The presence of morbid growths.

1. **Physical conditions.**—These present great variety and may either affect the whole muscle or only one lateral half. Of such as originate in the thorax and produce *downward* displacement, the more important are pneumothorax, pleural effusion accompanied by positive intrathoracic pressure, emphysema, pericardial effusion, extensive enlargement of the heart, and the presence of an intrathoracic growth. *Upward* displacement may be produced by contracting lesions originating within the lungs or pleura and by collapse of one or both lower lobes.

The abdominal conditions leading to *upward* displacement are pregnancy, distension of the stomach or intestines, ascites, peritonitis followed by intestinal distension or due to perforation of the stomach or intestines and accompanied by the escape of air into the peritoneal cavity, also marked enlargement of the liver from the presence of an abscess, hydatid tumour, or a new growth. A hydatid tumour or a new growth in the kidney or spleen and tumours of the uterus, ovaries, and other organs, frequently cause displacement in an upward direction. A similar change may result from the presence of a sub-diaphragmatic abscess.

Symptoms.—In many of the conditions enumerated the symptoms are chiefly those of the original disease and do not therefore need detailed description.

Enlargement of the abdomen produces a sense of tightness about the lower part of the chest, and, by interference with the action of the diaphragm, causes difficulty in breathing. If the abdominal distension is extreme and sufficiently long continued, collapse of the inferior parts of the lower lobes of both lungs results from their non-expansion, and thus the dyspnœa is increased.

The *physical signs* which characterise collapse of the bases of the lungs are diminution or absence of expansion of the lower lobes, inspiratory recession of the lower interspaces, and weak or absent breath sounds. If, as is frequently the case, some degree of œdema is also present, fine crackling râles will be audible at the bases. The level of the diaphragm may be determined by the aid of percussion and auscultation.

2. **Functional disorders.**—**Spasm**, which may be either *tonic* or *clonic*, may result from lesions affecting the phrenic nerves, either at their origin or in their course, or spasm may be due to direct irritation of the diaphragm, or it may arise from reflex irritation.

In tetanus, poisoning by strychnine, and in hydrophobia the spasm is tonic. Some writers have held that asthma is, or may be in some cases, due to tonic spasm of the diaphragm, but this view has not met with general acceptance. Tonic spasm has also been known to occur from excessive laughter, and it is a matter of common experience that severe pain in the region of the diaphragm may be thus induced. The diaphragm is sometimes thus affected in hysteria.

Symptoms.—Tonic spasm gives rise to pain and a sense of contraction of the chest. If continued for a sufficient time it is followed by almost complete cessation of respiration and by all the phenomena of asphyxia.

Hiccough is the most familiar result of clonic spasm. The sound is produced in the larynx by the inrush of air which follows the sudden descent of the diaphragm, the glottis being, so to speak, taken unawares in a condition of incomplete dilatation.

3. **Paralysis.**—Paralysis of the diaphragm is observed in diphtheria, and then almost invariably occurs in association with other paralyses due to lesions of the peripheral nerves. Paralysis from lead poisoning is also the result of peripheral neuritis. Paralysis of the diaphragm may also occur in Acute Ascending Paralysis (Landry's Paralysis), and in lesions of the cord due to injury or disease of the vertebræ. Hæmorrhage into the cord, or the presence of a tumour, at or above the site of origin of the phrenic nerves, may also give rise to this condition.

The *signs and symptoms of paralysis* of the diaphragm are:

1. Reversal of the respiratory movements of the epigastrium and hypochondria. These regions recede during inspiration instead of bulging.
2. Absence of downward movement of the abdominal viscera during inspiration.
3. Increased movement of the lower ribs.
4. Dyspnœa on exertion or excitement.
5. Alteration in the character of the voice and cough.
6. Loss of the compressive action of the abdominal muscles upon the contained viscera which ordinarily attends such acts as defæcation and the commencement (but not the continuance) of urination.
7. Feebleness of expiration and of such reflex expiratory actions as cough, sneezing, expectorating, are indirect results of paralysis of the diaphragm; as the presence of that condition prevents the previous inspiration from being sufficiently full to be followed by a forcible expiratory act.
8. Diminution of the total capacity of the thorax, owing to increased arching of the diaphragm.

Paralysis in diphtheria.—The condition in diphtheria has been carefully studied by Dr. W. Pasteur,[1] to whose valuable paper the writer is much indebted. It has necessarily been observed most often in children, owing to the greater frequency of that disease in early life.

Increased movement of the lower ribs was present in ten out of fourteen cases of that nature. Cessation or reversal of the normal movements at the epigastrium during respiration was observed in twelve cases. Alteration in the character of the cough and voice was noted in ten cases, but was probably present in all.

---

[1] 'Respiratory Paralysis after Diphtheria as a cause of Pulmonary Complications.' *International Journal of the Medical Sciences*: September 1890.

The following is an abstract from the notes of a case of paralysis of the diaphragm in diphtheria: A boy, æt. 4 years, was admitted to the North-Eastern Hospital for Children, under Dr. Pasteur, on August 18, 1888. He had been attacked by diphtheria in the middle of July, and paralysis of the palate was first noticed on August 17. Paralysis of the right leg appeared on August 20. The right arm was affected on August 22 and the left leg on the 28th. The following note was made on August 30:

'There is marked pallor and a somewhat earthy tint of the integuments. The breathing is deep, rather slow, and entirely thoracic. The voice is nasal and indistinct, and the child appears unable to cough. The epigastrium falls in during the deepest inspiration, and no downward movement of the abdominal viscera can be detected. The depression at the epigastrium fills up again during expiration. Paralysis of the diaphragm is apparently complete. All the intercostal muscles appear to act strongly, air enters well into the upper part of the lungs, but very imperfectly along the anterior margins and at the extreme bases. There are no râles.'

On Sept. 6, consolidation of the right base with moist râles appeared, and the temperature rose to 103° F., the respirations to 60 per minute, and the pulse to 170. There was gradually increasing asphyxia, and death occurred in the evening of that day—*i.e.* the twentieth day from the onset of the first sign of paralysis.

On post-mortem examination, the heart was uncovered by lung and the diaphragm was on a level with the third intercostal space. The whole of the right lower lobe was soft, wet, of a dark blue colour, and entirely devoid of air. On section lighter patches of broncho-pneumonia were seen around the smaller bronchi. The left lower lobe presented exactly similar changes, but they were less advanced. The posterior part of the right upper lobe was consolidated from recent pneumonia; it contained but little air, and presented a granular appearance on section. The remainder of the lobe was œdematous but crepitant. The left upper lobe was œdematous.

In the chapter on Collapse of the Lungs (*vide* p. 288) it is pointed out that failure of any part of the chest wall to expand during inspiration, if sufficiently long continued, is necessarily followed by collapse of the subjacent lung. The diaphragm stands in the same relation to the lower surfaces of the lungs as the chest wall does to other portions of their surface; and there can be no doubt that the collapse of the bases which accompanies paralysis of the diaphragm is the direct result of that condition. Feeble circulation, the result of weak cardiac action, may have some share in the production of œdema, but could not give rise to extensive collapse.

Diagnosis.—Increased horizontal expansion of the lower thoracic zone, with reversed movements and recession at the epigastrium, are typical signs of diaphragmatic paralysis. The increased movement of the lower ribs was often the earliest sign, and also the one most commonly present in the cases described in the paper already quoted. It is believed by Dr. Pasteur to be mainly due to the diminution or removal of the opposing or steadying action of the diaphragm on the lower ribs. The occurrence of extensive pulmonary collapse under these conditions, though at first sight somewhat paradoxical, is fully accounted for by the total loss of vertical expansion of the chest cavity. Cases of broncho-pneumonia with collapse of the lower lobes, to which those now under consideration may present a close superficial resemblance, are to be distinguished by the recession and diminished movements of the lower ribs and bulging epigastrium. In broncho-pneumonia with collapse of the lower lobes, the condition which most nearly resembles paralysis of the diaphragm, the signs observed are reversed, as in such cases the movement of the lower ribs is less than normal and the diaphragm bulges downwards.

Prognosis.—This is under all circumstances grave, but in cases of paralysis due to peripheral neuritis, as in diphtheria, recovery may follow, even when collapse of one lower lobe has occurred; when both are involved the outlook is very unfavourable.

In cases of paralysis of the diaphragm of spinal origin the prognosis necessarily depends in some measure upon the exact nature of the lesion, but such a complication is invariably of serious import and often leads to a fatal termination.

In acute ascending paralysis, if the diaphragm is paralysed and respiration is seriously affected, recovery is rare.

Treatment.—Artificial respiration has proved of great service in cases of paralysis due to diphtheria. It may be employed for ten minutes every four hours, or oftener, if the symptoms are very urgent, and may be combined with the use of hypodermic injections of brandy and ether. The continuous inhalation of oxygen, warmed during its passage through a metal coil placed in a vessel containing hot water, is also to be recommended.

Similar methods of treatment are indicated in all cases of paralysis due to lesions of the phrenic nerves. If only one half of the muscle is involved the patient should be made to lie upon the opposite side in order to facilitate the expansion of the affected lung. By the aid of pressure applied to the lower part of the chest the act of coughing may be assisted. The best evidence of improvement is the free entry of air into parts of the lung where previously breath sounds were absent. In cases of spinal origin the treatment must necessarily depend to a great extent upon the nature of the primary morbid condition, but by the aid of artificial respiration and stimulants it is possible that life may be prolonged after paralysis of the diaphragm has occurred.

DIAPHRAGMATIC PLEURISY

When the pleura covering the diaphragm is primarily involved and the inflammatory process is acute, the accompanying symptoms may be of a most severe character.

Symptoms.—The exact interpretation of these symptoms is, however, rarely free from difficulty, owing to the fact that on physical examination few signs of pleurisy, if any, may be discoverable. The most striking symptom is pain, of a severity not observed in any other form of pleurisy.

This may be referred to the epigastric or hypochondriac region, or to the line of the tenth rib and along the insertion of the diaphragm, and tenderness may also be present over the same areas. The patient generally sits up in bed and may lean forward, but usually varies his position from time to time. His face is marked by great anxiety and the degree of suffering is extreme.

The breathing is rapid; expansion on the affected side is limited to the upper part of the chest, and is in excess on the opposite side. The diaphragm is fixed but not paralysed, and the abdomen does not move. The temperature is high, and in some cases delirium has been observed. Hiccough, vomiting, and dysphagia may also be present.

No friction sound may be audible during any period of the disease, but signs of effusion may be found later, and their appearance may coincide with a relief from the most severe symptoms. In the majority of cases, however, there is no effusion, or, if it occurs, the quantity is insufficient to produce definite signs.

Diagnosis.—The symptoms are nearly always suggestive of some acute affection of the abdomen, but nothing can be discovered there to account for them, and, if the type of breathing is as above described, the inference is that the case is one of diaphragmatic pleurisy. The disease is rare, but we have met with cases which were almost certainly examples of it, and in which the clinical picture was typical. As recovery followed without the development of any other affection, it is very probable that the diagnosis was correct.

It is, however, certain that a pleurisy may extend to the surface of the diaphragm without giving rise to any distinctive clinical characters; otherwise, having regard to the frequency with which old and recent lesions are found post mortem in this situation, the group of symptoms which accompany a primary pleurisy in that region should be far more common.

Prognosis.—The danger to life is probably not nearly so great as the condition of the patient and his fears suggest; but death may occur without any other lesion being present. In the great majority of cases recovery follows after an illness which is usually of short duration, unless effusion occurs.

Treatment.—The immediate indications for treatment in severe cases are to relieve the pain and mental distress, and both these are fulfilled by giving a hypodermic injection of morphia. Local applications are also necessary; strapping the affected side may be successful, but, if the patient is unable to bear pressure, hot opium fomentations or poultices should be used, or leeches may be first applied and subsequently poultices.

J. K. F.

CHAPTER LXII

PAIN IN DISEASES OF THE LUNGS AND PLEURA

THE first attempt to explain the association of pain in certain regions of the body with disease situated at a distance from the site of the pain is contained in the classical lectures of John Hilton on 'The Influence of Rest in the Treatment of Accidents and Surgical Diseases and the diagnostic value of Pain.'

'Pain in any part, when not associated with increase of temperature (the local symptom of local inflammation), must be looked upon as caused by an exalted sensitiveness of the nerves of the part and as a pain depending upon a cause situated remotely from the part where it is felt. These so-called sympathetic pains . . . result from some direct nervous communication passing between the part where the pains are expressed and the real and remotely situated cause of the pain' (Hilton, p. 72).

The work which Hilton commenced has been completed and extended with conspicuous ability by Dr. Henry Head,[1] to whose papers the writer desires to acknowledge his indebtedness for the following account of 'referred' pain and tenderness which may accompany diseases of the lungs and pleura.

'Two quite distinct varieties of pain, accompanied by two even more distinct types of tenderness, may make their appearance in the course of diseases of the lungs and pleura.

'Pain of the first type is *local*, and occupies no predetermined spots or areas. It is situated over the focus of disease, and usually in those intercostal spaces which cover a patch of pleural friction. It is accompanied by no true superficial tenderness, but marked deep tenderness is evoked by pressure or percussion.

'Pain of the second type is *referred*—that is to say, it is not necessarily situated over the focus of disease. It runs through or round the body, and has a focus behind and one in front. If severe or of any duration, it is accompanied by more or less marked tenderness of the superficial structures of the chest, and this tenderness lies over predetermined spots or bands. Thus referred pain and tenderness do not point directly but only indirectly to the focus of disease' (Head, *op. cit.* p. 231).

---

[1] *Brain*, Parts lxxiv. and lxxv.

Local pain.—This is observed in cases of acute pleurisy, pneumonia with pleurisy, also in pulmonary tuberculosis. We may remark incidentally that a complaint of pain at the upper part of the chest, about the apex of the lung, should always lead to a careful examination of the lungs, as it is often present at a very early period of the disease.

The pain in pleurisy is generally accompanied by tenderness on pressure over the site of the disease. Dr. Head cites cases showing the value of tenderness on deep percussion in localising the site of pulmonary infarctions situated upon the surface of the lung.

Referred pain.—This variety of pain has no localising value; it is situated at certain spots on the chest, or may appear to run round the body at various levels. 'In these spots we recognise the maximum points of certain areas connected with the central arrangement of the nervous system' (Head).

Pain of this character is associated with tenderness of the superficial structures of the chest wall, and is often observed in cases of pulmonary tuberculosis. It is relieved by 'firm pressure over a broad surface' (Walshe).

Tenderness may be present over extensive areas in such cases, and, if the skin be raised between the finger and thumb, complaint is made of pain.

'In cases of costal pleurisy of the lower half of the chest, it is a common occurrence that the upper half of the abdominal parietes is tightly drawn backwards, and the skin over that part very tender to the touch; and that pain, depending upon pleurisy of the lower half of the chest, is referred to this spot. Here also we have an exemplification of the same nerve supplying the muscles, the skin over the muscles, and the peritoneum associated with those muscles' (Hilton).

Hilton quotes an interesting case of double pleural effusion accompanied by symmetrical pain referred to the same site, and described by the patient as being 'on both sides alike.' The writer has observed several cases of pleurisy in which the pain was referred to the opposite side of the chest.

The practical outcome of these observations is that whenever a patient complains of pain, particularly if it is referred to the epigastric region, the chest should be carefully examined for signs of pleurisy, not on one side only, but on both.

Dr. Head has found that referred pain and tenderness may be associated with areas of referred pain and tenderness on the scalp, showing that both are dependent upon impulses passing from the central nervous system. Pain of this character is 'produced by distension of a sensitive organ from within, or by the exertion upon it of some tearing or rending strain from without.' In cases of pulmonary tuberculosis, the appearance of referred pain and tenderness may coincide with an extension of the disease; in other cases it may be absent throughout. Both are most marked in cases associated with bronchitis. For a detailed account of the attempt to connect the presence of certain areas of superficial tenderness with disease of certain portions of the lung, we are compelled to refer the reader to Dr. Head's paper. It is evidently a task of some difficulty, and is attended with many sources of fallacy to avoid which requires experience.

Dr. Head's conclusions on the subject of referred pain are as follows:

1. All referred pain and superficial tenderness which make their appearance from whatever cause in the course of subacute phthisis tend to spread widely. This is probably due to the cachexia and pyrexia which accompany the disease.
2. Referred pain produced by disease of the lung is most marked and of greatest intensity on the same side of the trunk as the fresh lesion. Both pain and tenderness may, however, appear secondarily on the side opposite to the lung affected, but they appear later and disappear earlier, and are not of the same intensity as when the side of the body affected corresponds with that of the lung which is diseased.
3. The innervation of the lung is connected with the third and fourth cervical segmental areas and with all the dorsal segmental areas from the third to the ninth. The lower lobes of the lungs are particularly connected with the dorsal areas, especially from the fifth dorsal area to the ninth.

J. K. F.

CHAPTER LXIII

TUMOURS OF THE MEDIASTINUM

PAGE
Primary tumours 675
Varieties 675
  The term 'lympho-sarcoma' 677
Site of the growth 677
Side affected 678
Causation 678
Age 678
Sex 678
Primary seat of mediastinal growths 678
Pressure effects 679
  Lesions of the sympathetic nerves 680
Morbid anatomy 682
Secondary tumours 683
Associated lesions 683
Symptoms 684
Symptoms of secondary growths 687
Physical signs 687
Course and duration 688
Prognosis 689
Diagnosis 689
Diagnosis of the nature of the growth 693
Treatment 693

PRIMARY TUMOURS

In considering the subject of tumours of the mediastinum, we shall deal chiefly with those forms of tumour, viz. sarcoma and cancer, which are of most common occurrence, and shall reserve for separate consideration the varieties of morbid growths, such as lymphadenoma, fibroma, dermoid tumours and hydatid cysts, which are but rarely met with.

Varieties of tumours.—Some difference of opinion exists as to the kind of tumour which is of most common occurrence. By the majority of pathologists this is believed to be sarcoma, but the collection of a number of recorded cases from various sources appears to show that a larger proportion of these growths are cancers.

Hare[1] has collected 520 cases of disease of the mediastinum which are classified thus:

	Cases
Cancer	134
Sarcoma	98
Mediastinal abscess	115
Lymphoma Lymphadenoma	21
Non-suppurative inflammation	16
Fibroma	7
Hæmatoma	6
Dermoid cysts	11
Hydatid cysts	8
Miscellaneous	104
	520

The 104 cases classed as 'miscellaneous' include malignant growths of uncertain nature, tumours rarely met with in this region, such as enchondroma, osteo-sarcoma, chondroma and lipoma, enlarged glands, chiefly tubercular, cases of fibroid thickening, gunshot wounds, foreign bodies and of mediastinal emphysema.

In the reports of 1,800 autopsies made at the Brompton Hospital, we have found thirty cases of mediastinal tumour in which the nature of the growth is thus stated:

	Cases
Round-celled sarcoma	9
Large-celled sarcoma	1
Lympho-sarcoma	7
Cancer	7
Columnar epithelioma	1
Lymphadenoma	2
Not expressly stated	3
	30

Of these cases it may be remarked that where the report of the microscopical examination is most complete the growth generally proves to be a round-celled sarcoma. Notwithstanding the table given by Hare, we are still disposed to believe that sarcoma is the growth of most frequent occurrence—a view which is in accord with the statement of Fagge, that 'the vast majority of them are made up principally or entirely of small round cells, and are classified either as lymphomata or round-celled sarcomata.'[2] Dr. Steven,[3] in discussing Hare's cases, agrees with the view here expressed, and points out that of the 134 cases of cancer contained therein sixty-seven are recorded in or prior to the year 1870, at a time, that is, when many growths were called cancer, which would now be otherwise classified, whereas of the ninety-eight cases of sarcoma only seven belong to the same period.

It is also significant that of the 134 cases of cancer collected by Hare, in only forty-seven are the microscopical characters of the growth so definitely stated as to allow of their classification. Of these thirty-two were medullary (encephaloid), thirteen scirrhus, and two colloid. Another fact tending to invalidate this evidence is that in fifty-six of these cases of tumour no mention is made of the seat of the primary growth.

---

[1] Fothergillian Prize Essay. Medical Society, London: 1889.

[2] *Op. cit.*, vol. ii. p. 115.

[3] *The Pathology of Mediastinal Tumours.* London: 1892.

It is necessary at the outset to define clearly the meaning attached to the term '**lympho-sarcoma.**' It is variously used:

(*a*) To signify a growth of a sarcomatous nature originating in a lymph gland.

(*b*) To indicate the presence in a round-celled sarcoma of a fine reticulum similar to that found in a lymph gland.

(*c*) As synonymous with lymphadenoma.

Dr. Steven considers that the histological basis is too narrow for purposes of classification, and, although admitting that these growths are really round-celled sarcomata, prefers to call them lympho-sarcomata because they originate in lymph glands.

The writer is unable to accept this view, and considers that the term should be restricted to sarcomata, consisting of rounded or 'lymphoid' cells in which a fine reticulum is present, similar to that found in a lymphatic gland.

It is impossible here to consider fully the difficult question of the relationship of various glandular enlargements to the disease which Hodgkin described, but whilst admitting the occasional origin in the mediastinal glands of the general glandular enlargement present in that affection, we hold that neither clinically nor pathologically is there such a close relationship between lympho-sarcoma of the mediastinal glands and lymphadenoma as to justify the use of the two terms as synonyms.

Site of the growth.—In the cases collected by Hare this was as follows:

	Cancer	Sarcoma
Anterior mediastinum alone	48	33
Posterior „ „	20	8
Entire „	8	8
Anterior and middle alone	8	1
Anterior and posterior alone	14	3
'Whole thorax'	2	1
Middle mediastinum alone	5	3
Posterior and middle „	2	—

It would therefore appear that, without including the cases in which a growth originating in the anterior mediastinum extends to the middle mediastinum, the anterior is affected nearly three times as often as the posterior.

This conclusion is opposed to the view which we believe to be held by many observers in this country, viz. that the posterior mediastinum is the most common site of origin of growths in this region of the body.

There is, however, a considerable amount of evidence in support of Hare's conclusion. Wilson Fox states that the anterior mediastinum is affected nearly twice as often as the posterior.

Pepper and Strengel[1] give the relative frequency of primary tumours in the anterior mediastinum as nearly three or four to one.

Dr. V. D. Harris[2] records nine cases of primary malignant disease of this region, of which eight were in the anterior mediastinum and one in the posterior, and of a similar number of cases examined by Dr. H. D. Rolleston,[3] six were primary in the anterior space and three in the posterior.

It is often very difficult at a post-mortem examination, owing to the wide extent of the growth, to determine in which part of the mediastinum it commenced, and it is still more so from reading post-mortem reports which do not contain an absolute statement on the point.

Side affected.—The growth rarely involves both sides of the chest equally, and when limited to one side, the right is far more often affected than the left. Thus, in thirty cases which we have analysed, the right lung was infiltrated in twenty, the left in five, and both lungs in five.

When both lungs are involved, the right is, as a rule, more extensively implicated than the left.

Causation.—No definite statement is possible as to the cause of primary malignant growths in the mediastinum.

Age.—A considerable proportion of cases of mediastinal tumour occurs below the age of 40 years. In thirty cases analysed by the writer the age distribution was as follows:

Age	1-10	11-20	21-30	31-40	41-50	51-60	over 60
No. of cases	1	2	8	6	10	3	0

The average age at death in twenty-four cases in males was 36 years, and in four cases in females 44 years. Hare gives the average age in cancerous growths as males 37·7 years, females 36 years, and in sarcoma, males 30–35 years, females 35–40 years.

Sex.—The male sex predominated remarkably in the Brompton Hospital cases, viz. twenty-five males, five females. This proportion is considerably larger than that given by other observers for cases of mediastinal tumour (Riegel 2·4 to 1; Köhler 5 to 3; Hare 2 to 1). Walshe states that in cancer the sexes are about equally affected (102 cases: males 58; females 44).

PRIMARY SEAT OF MEDIASTINAL TUMOURS

The origin of cancer from epithelial structures and of sarcoma from those of the connective-tissue type is a well-established fact in pathology, and must be true of malignant growths occurring in the mediastinum as elsewhere.

---

[1] *Trans. Assoc. American Physicians*, 1895: vol. x. p. 202.

[2] *St. Bartholomew's Hospital Reports*, 1892: vol. xxviii. p. 73.

[3] *Journal of Path. and Bact.*: December 1896.

Anterior mediastinum.—Tumours in this situation often originate in the thymus gland, and may present the structure either of cancer or sarcoma. Dr Rolleston[1] has described an interesting case of hæmorrhagic adeno-chondro-sarcoma which appeared to have originated from a diverticulum of the thymus gland. A sarcoma may originate from the mediastinal connective tissue or lymph glands or from the periosteum of the sternum or the subpleural connective tissue. Enchondroma may arise from the cartilages of the ribs, and syphilitic gummata may project from the posterior aspect of the manubrum sterni and compress the subjacent structures. Dermoid cysts may also be present in this situation, and the thyroid gland may possibly be the site of origin of a tumour of the anterior mediastinum.

Middle mediastinum.—Growths probably originate in this division of the mediastinum less often than in either of the others. Sarcomata may arise from the subpleural connective tissue of the pericardium and the tunica adventitia of the large blood-vessels.

Posterior mediastinum.—Sarcomata and lympho-sarcomata commonly originate from the tracheal and bronchial lymph glands. Cancer, which is more often found in this division of the mediastinum than elsewhere, may arise in the epithelial structures of the trachea, bronchi and œsophagus.

Sarcomata may also arise from the periosteum of the vertebræ.

PRESSURE EFFECTS

Two varieties of pressure may be recognised—the one *centrifugal*, in which the effects are due to a force acting from the centre towards the periphery; the other *centripetal*, in which the organs within the thorax are compressed by a constricting force acting towards the centre.

The structures subjected to pressure necessarily vary according to the site of the tumour, and the effects observed depend upon the degree to which the organs are compressed.

Anterior mediastinum.—A growth originating in this region may erode the sternum and ribs or the clavicle, and may perforate the chest wall. The glands in the posterior triangles of the neck frequently undergo enlargement. Tumours in the anterior mediastinum tend to spread backwards, and, as a rule, the large vessels become involved at an early period of the disease.

Middle mediastinum.—The structures in the middle mediastinum may be involved primarily, or, as the growth extends, either from the anterior or posterior mediastinum. Narrowing of the superior vena cava is at first attended by enlargement of the innominate, subclavian, and jugular veins, and later by œdema of the face, neck, thorax, and arms. When the vessel is completely occluded there may be enormous dilatation and marked tortuosity of the superficial vessels of the thorax, which may empty their blood into almost equally enlarged epigastric veins. It is in such a case generally easy to demonstrate that the flow of the blood is from above downwards.

---

[1] *Journal of Pathology and Bacteriology*, December 1896.

Compression of the *inferior vena cava* may be attended by œdema of the lower extremities and ascites. In a case of this kind under the care of the writer, the legs were almost black from dilatation of the vessels of the skin, but for a long period there was no œdema.

The *aorta* or one of its main branches may be compressed; in the latter case there may be either retardation or a difference in volume of the pulse in the peripheral arteries. Some degree of displacement of the *heart* almost invariably results from the presence of a mediastinal growth, the direction varying with its position; downward displacement is by no means uncommon.

The auricles may be encroached upon by the growth and the mitral orifice narrowed. If the pulmonary artery is surrounded and stenosed, great hypertrophy and dilatation of the right ventricle may result. If one of the main branches of the vessel is compressed, a thrombus may form within it, and in the corresponding lung numerous infarctions may be found. Infiltration of the pericardium may set up acute pericarditis or a blood-stained serous effusion may occupy the cavity.

The phrenic nerves may be involved in the growth, and downward displacement of the *diaphragm* frequently occurs. This, however, is rarely the result of impaired innervation.

Posterior mediastinum.—The *trachea* may be narrowed and one of the main bronchi or the large branches distributed to the part of the lung most involved may be obliterated, as the growth commonly extends into the lung along the bronchi and large vessels. Perforation of the bronchi is not uncommon.

The œsophagus may be compressed and dysphagia may be present. There may also be pressure upon the thoracic aorta, and azygos veins, in the later case leading to enlargement of the intercostal veins and possibly to œdema of the chest wall. In cases in which marked emaciation has been observed, this has been attributed to compression of the *thoracic duct*. One or both *pneumogastric nerves* may pass through the growth or may be flattened by it, and the *recurrent* branch of the left vagus may be involved. Pressure upon both recurrent branches may be followed by bilateral paralysis of the abductors of the larynx, and pressure on the left recurrent laryngeal nerve by paralysis of the abductors of that side.

Paralysis of the sympathetic nerve may give rise to various changes in the eye.

If the sympathetic is paralysed, the *size of the ocular slit* is diminished, not by a simple dropping of the upper lid, as in ptosis from weakness of the levator palpebræ superioris, but from an imperfect opening of both lids. This depends upon paralysis, partial or complete, of the unstriped muscular fibres in the lids, described by

Müller, which are controlled by the sympathetic. Most of these fibres, if not all, pass vertically and are inserted into the margins of the lids, but some are said by Henle to pass horizontally. The eyelids are, of course, not motionless, but the eye cannot be opened so widely as in the normal state.

The eyeball appears to have receded.—This is no doubt to some extent only apparent and results from the partial closure of the lids. But there is also some actual recession, depending upon the paralysis of the other unstriped muscular fibres described by Müller, which bridge across the sphenomaxillary fissure. The contraction of these fibres causes the eyeball to protrude.

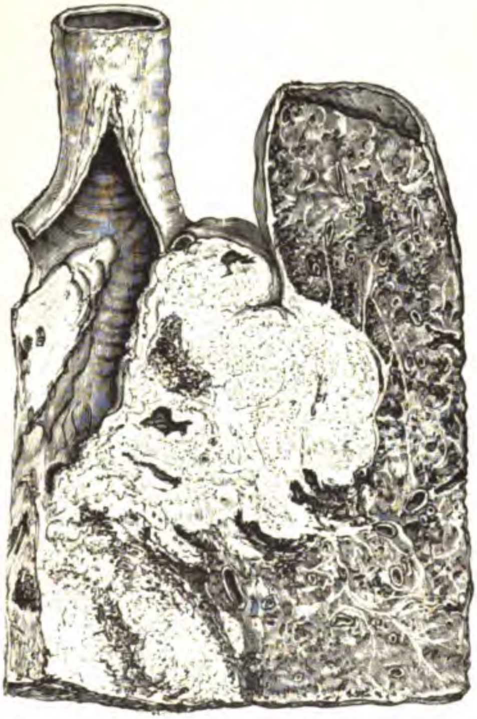


FIG. 149.—PORTION OF A MEDIASTINAL LYMPHO-SARCOMA, TO SHOW THE MANNER IN WHICH THE TUMOUR EXTENDS ALONG THE BRONCHI AND PULMONARY VESSELS

(From Mr. J. Bland Sutton's work on Tumours.)

The pupil is smaller than that on the opposite side; or it should rather be said it does not dilate as widely as that on the healthy side. It may thus happen that the pupils, when exposed to a full light, are equal, but when shaded that on the affected side is the smaller of the two.

The dilator fibres of the pupil arise from the first, second, and third dorsal nerves (some say also the seventh and eighth cervical) and pass upwards in the ascending branch of the superior cervical ganglion; the motor fibres of the involuntary muscles of the orbit and eyelids come from the highest four or five dorsal nerves, and thus an affection of the upper part of the chest may produce the results we are describing. It is theoretically possible that the diminution of the ocular slit and recession of the eyeball might occur without any affection of the pupil.

Unilateral sweating has also been observed in cases of mediastinal tumour accompanied by pressure upon the sympathetic nerve.

Irritation of the sympathetic is a much rarer event, and gives rise to conditions the opposite of those above described.

The importance of observing these changes in the eye is not so much in connection with obvious cases of aneurysm or mediastinal tumour, as in those not uncommon ones where a pleural effusion is not easily accounted for. The presence of the ocular symptoms will suggest the existence of some mediastinal tumour, which need not, however, be malignant. They may result, as has been said, from the pressure of an aneurysm or enlarged mediastinal glands; and, though we can bring no cases verified by a post-mortem examination to justify our belief, we think it not improbable that the contraction of inflammatory products in the neighbourhood of the apex of the lung may, in exceptional circumstances, produce the same result.

It is remarkable that these very obvious phenomena do not appear to have been observed before 1850, and to have been first described in this country in 1855 by Dr. Walshe; but even now slight degrees of the affection are, we believe, not infrequently overlooked.

MORBID ANATOMY

Sarcomata and lymphosarcomata usually form tumours of large size which may occupy the greater part of the upper portion of the thorax.

In the posterior mediastinum the growth may surround the bifurcation of the trachea and perhaps occlude the main bronchus of one lung and extend along its branches and those of the pulmonary vessels far into the interior of the organ, extensive areas of which it may replace. As it extends further the growth may invade the pericardium and surround the large vessels and form a considerable mass in the anterior mediastinum or in the triangles of the neck. In some cases the growth extends along the pleura and surrounds the lung with a thick capsule, from which prolongations extend into the interlobar fissures. A similar growth originating in the anterior mediastinum may form a large mass in one or both sides of the chest, and extend downwards beneath the sternum and backwards around the large vessels and the heart.

Sarcomata and lymphosarcomata usually appear as soft, white or pinkish, fleshy looking lobulated masses, in which areas of hæmorrhage or fatty degeneration may be present, or they may be of a denser and almost fibrous consistence, and present a well-defined outline limited by a capsule from which trabeculæ extend into the growth. In their early stages the softer growths are also usually well defined.

Cancers usually form smaller nodulated growths of firmer consistence, which tend to undergo ulceration and form cavities within the lungs more often than sarcomata. Probably for the same reason the presence of a cancerous growth more often excites inflammatory changes in the lung and is more frequently associated with gangrene.

Secondary tumours of the mediastinum.—After the removal of a primary sarcoma, or in cases in which, although such a growth is present, no operation has beeen performed, a secondary tumour may occur in the mediastinum, but the lungs are more often the site of metastasis or reappearance of such growths. When the mediastinum is the site of a secondary growth the tumour may be of large size. As an example of this we may cite a case in which a very small melanotic sarcoma of the foot in a young woman was followed by a secondary growth which occupied almost the whole of the mediastinum, and gave rise to signs which for a time were thought to be due to the presence of an empyema.

Secondary cancerous deposits may occur in the mediastinal glands from the direct extension of a growth of the same nature in the œsophagus, or by infection of the prevertebral glands either from a primary growth in the abdomen or peritoneum, or in the neck.

In cancer of the breast, if operation is long delayed and the chest wall or pleura becomes infected, a secondary deposit is often present in the mediastinal glands.

ASSOCIATED LESIONS

Bronchi and lungs.—The bronchi may be extensively inflamed and filled with frothy fluid, or there may be saccular ectasia beyond the site of narrowing. In some cases bronchiectasis is so extreme that the greater part of a lung is converted into smooth-walled cavities. Such cavities may be filled with fœtid secretion or the dilated bronchi may be found leading into ragged or gangrenous cavities in the lungs. The contents of such cavities may be both puriform and fœtid.

Almost a whole lung may be thus destroyed, and the abscess which results may perforate an interspace and form a collection of pus external to the chest. In some cases it has been mistaken for an empyema and tapped. The occurrence of destructive and inflammatory changes in the lungs, in cases of mediastinal tumour in which the pneumogastric nerves are involved, has been attributed to disturbed innervation, but the evidence in favour of this view is not convincing.

Cavities may be formed by the breaking down of nodules of the growth, and the portions of the growth remaining may form the walls of the cavities and surround the obliterated bronchi and vessels which cross their interior.

Some degree of collapse of the lung commonly occurs; in some cases it is the result of complete obstruction of a bronchus, in others it is due to the presence of a pleural effusion.

The lungs are often emphysematous and œdema is rarely completely absent. Inflammatory consolidation is common, especially in cases of cancer, and fibrosis and reticular cirrhosis may also be present. There may be extensive hæmorrhages or infarctions secondary to thrombosis or embolism, and thrombi are frequently found in the narrowed branches of the pulmonary artery.

Secondary growths in other organs.—In the 30 cases which we have analysed secondary growths were found in the following sites:—Liver 6; pancreas 6; supra-renals 3; spleen 3; kidneys 2; heart 2; brain 2; omentum and mesentery 3; and in the thyroid gland 1. Secondary infiltration of the chain of glands lying along the spine and extending into the pelvis is frequently observed.

A growth originating in the head of the pancreas, and subsequently involving the mediastinal glands, may cause jaundice from compression of the common bile duct, or jaundice may be present in a case of mediastinal tumour from compression of the duct by a mass of enlarged glands in the transverse fissure of the liver. The liver may be enormously enlarged; in one of the above cases it weighed 7 lb. 12 oz.

Tuberculosis.—In two of the above cases tuberculosis was present in association with a malignant growth. The co-existence of active pulmonary tuberculosis and mediastinal tumour has been doubted, and it is certainly of rare occurrence. In one of the cases examined in which a malignant growth, probably originating from the periosteum of the vertebræ, infiltrated the posterior mediastinum, large irregular tubercular cavities were found in the lower lobe of the left lung and the upper lobe of the right lung, and some dry caseous nodules were present in the same situations. The pus in the cavities contained masses of tubercle bacilli. In another case the lesion consisted of pigmented fibroid nodules in both lungs.

SYMPTOMS

The symptoms produced by mediastinal tumours depend to some extent upon the nature of the growth, whether sarcoma or cancer, but from a practical point of view this is of so little importance that description of the clinical features peculiar to each may be reserved for the section on diagnosis.

The onset of the disease is, as a rule, insidious, cough with ex-

pectoration, pain in the chest, shortness of breath, and some emaciation being the most common symptoms at an early stage.

Hæmoptysis occurs in the majority of cases; it may be profuse and frequently repeated, and may appear before any other symptom.

Pleurisy, which is often accompanied by effusion, may precede any symptoms pointing to involvement of the mediastinum or lungs. In some cases the early symptoms are those of bronchial catarrh, or they may suggest the presence of pulmonary tuberculosis.

When the growth originates in, or chiefly involves, the anterior mediastinum, signs of pressure upon the venous trunks appear early; or the superficial veins of the neck and thorax or of one arm are enlarged, there may be lividity of the face, and prominence of the eyes, and at a later period the congested parts may become œdematous. These signs may be limited to, or most marked on, one side. Pain is often localised beneath the sternum and may be accompanied by tenderness. In a similar manner growths in the posterior mediastinum compress the trachea and bronchi or the œsophagus at an early period and give rise either to stridor or dysphagia. Pain in these cases may be felt in the back or between the shoulders, or may extend up the neck or shoot down the arms. Pressure upon the intercostal nerves may give rise to intense pain on one side of the chest, and this may be accompanied by herpes zoster.

Cough is rarely absent and is often paroxysmal in character. The special feature which it acquires when due to pressure upon the trachea or main bronchi, or to paralysis of the abductor muscles of the larynx, is a brassy or stridulous tone.

Expectoration, owing to the accompanying bronchitis, is usually present; it may be mucoid or mucopurulent or 'prune juice' in character, and is frequently blood-streaked. When narrowing of the bronchi has led to bronchiectasis, profuse fœtid expectoration may be the most marked feature of the case. If gangrene supervenes, the odour characteristic of that condition will be observed. When the growth is undergoing softening, portions sufficiently large to be recognised by the aid of the microscope may appear in the sputa.

Dyspnœa is one of the most marked symptoms of the affection. It is at first experienced on exertion or it may be most severe when the patient lies down. At a later period it may be accompanied by stridor. If the growth originates in the posterior mediastinum and compresses the trachea and main bronchi, it may give rise to paroxysmal dyspnœa at an early stage, and before any other symptoms appear. Dyspnœa may be due to a variety of other causes, such as œdema of the lung, pressure on the pulmonary veins, or if both recurrent laryngeal nerves are involved, a rare condition in these cases, to bilateral paralysis of the abductors of the larynx. As the growth extends and the respiratory area is diminished, the dyspnœa increases and orthopnœa appears. In some cases dyspnœa is due to collapse of the lung from complete obstruction of a main bronchus or to collapse from pleural effusion. Pressure upon one main branch of the pulmonary artery or the complete obliteration of a main branch, although necessarily obstructing or preventing the access of blood to the lung of the affected side, is rarely *per se* a cause of dyspnœa, as respiration can be carried on if not more than one-fourth of the normal quantity of blood is passing through the lungs.

The breathing is often laboured and of the wheezing asthmatic type, especially when the growth is situated posteriorly and compresses the trachea or main bronchi, and in such cases it may only acquire the stridulous character when the patient lies down. In some cases the respiration is merely quickened.

Pressure upon one or both pneumogastric nerves may give rise to attacks of paroxysmal cough and dyspnœa, and to attacks of palpitation with greatly increased frequency of the pulse, but in some cases marked slowing of the pulse has been observed. In one case where a tumour was present above the clavicle, pressure upon the tumour caused dilatation of the pupil and alteration in the frequency of the pulse, both retardation and quickening having been noted.[1]

Pressure upon the *phrenic* nerves has been known to cause spasm of the diaphragm.

Pyrexia.—Fever is by no means a necessary or a constant symptom, and in both cancer and sarcoma the temperature may be subnormal. In the majority of the cases which we have analysed, fever, although usually moderate in degree, was present at some period of the disease, and towards the close it was often of an irregular hectic type. The conflict of evidence which exists as to the temperature in cases of mediastinal tumour depends no doubt partly on the fact that pleurisy, pericarditis and other inflammatory conditions are frequent but not invariable complications. The pulmonary changes which are usually set up by mediastinal growths are by no means always the same, and this also accounts for great differences of temperature in cases which otherwise are closely allied.

Emaciation is generally present to some degree, but is not so marked as in pulmonary tuberculosis; it has, however, been known to progress rapidly. The condition of cachexia so often accompanying cancer in other organs is rarely present in cases of mediastinal tumour.

A variety of other symptoms such as anæmia, incapacity for exertion, loss of appetite, and indigestion may attend the condition, but none are characteristic.

Clubbing of the fingers and toes is not uncommonly met with.

Cases are on record in which a growth involving, but probably not originating in, the posterior mediastinum has infiltrated the vertebræ and caused paraplegia from extension to the spinal cord; but such an event is extremely rare.

Obstruction to the return of blood from the brain may give rise to headache, giddiness and drowsiness, epistaxis and exophthalmos; buzzing or roaring noises in the ears may also be present, caused in the same way.

---

[1] Rossbach, quoted by Fagge, vol. ii. p. 119.

Symptoms due to secondary growths.—The symptoms to which secondary growths may give rise do not differ from those already described, and in such cases a knowledge of the fact that the patient has undergone an operation for the removal of a tumour usually leads to a correct interpretation of the pulmonary symptoms.

It should be remembered that there may be a long interval, a year or two years or more, between the removal of the tumour and the appearance of the growth in the mediastinum.

PHYSICAL SIGNS

Owing to the great difficulty which attends the diagnosis of the affection a methodical investigation of the chest is specially necessary, and the prescribed order of examination should always be followed.

Inspection.- The condition of the face and neck (cyanosis, lividity, œdema), of the eyes (exophthalmos, congestion of the conjunctiva), of the superficial veins of the neck and thorax (enlargement, marked tortuosity), and of the arms (œdema) should be noted. The intercostal veins may be enlarged and tortuous. Careful search should be made for enlarged glands in the triangles of the neck or in the axillæ. Prominence of the manubrium or of the sternal ends of the clavicles may be observed, and distinct nodules of the new growth may possibly be seen in the subcutaneous tissue. Bulging of one side may be obvious, or retraction may be present; intercostal depression may be absent in certain spaces. During inspiration the expansion of one side, either as a whole or in a certain area only, may be diminished or absent, and that of the opposite side increased.

Palpation.—The cardiac apex may be displaced, according to the position of the growth and its effect upon the lung; a downward displacement is perhaps most common. A systolic impulse, which is heaving but not distensile, may be felt over a considerable area occupied by a growth, and may suggest the presence of an aneurysm. The liver may be felt below the costal margin, especially in cases of tumour chiefly involving the right lower lobe.

Over a growth situated immediately beneath the chest wall the vocal fremitus is generally absent; but where lung tissue with patent bronchi intervenes, the vocal fremitus may be retained or even increased.

Percussion.—Over tumours which are in actual contact with the chest wall—and therefore more commonly over those which occupy the anterior mediastinum—the percussion note is absolutely dull, and the sense of resistance is increased. If the growth overlies the trachea or a main bronchus the note may acquire a tubular character. Various modifications of the percussion note —viz. amphoric, tympanitic, or 'cracked pot'—may be produced by the intervention of lung tissue, in a state of consolidation, collapse, or emphysema, between the tumour and the chest wall. Growths in the posterior mediastinum, owing to their tendency to spread along the bronchi and vessels, do not so readily reach the chest wall; but when they have attained a considerable size and produced extensive changes in the lung, a dull note may be obtained over a large area.

Auscultation.—The respiratory sounds are absent over a solid growth which has occluded the bronchi; but when a tumour is situated between the trachea and the manubrium or in the posterior mediastinum, and compresses the trachea, loud bronchial breathing may be audible. Diminished expansion, from whatever cause arising, is necessarily attended by feebleness or absence of the vesicular breath sound; but if there is stridor that sound may be conducted through a solid mass. If exudation into the bronchi occurs, sonorous and sibilant rhonchi may be heard; also fine crackling râles due to œdema of the lung. Crepitant sounds are not uncommonly produced in the pleura in these cases. Pericardial friction may also be heard.

When a growth has undergone extensive softening, or has produced destructive changes in the lungs, cavernous breathing and pectoriloquy will be audible if the bronchi are patent. The heart sounds are often well conducted through a growth. A murmur may be heard, if the aorta or pulmonary artery is compressed, in such a manner as to give rise to the conditions necessary for its production—viz. a flow of blood through a narrow orifice into a wider space beyond; but a continuous narrowing of a vessel is not necessarily attended by the production of a murmur. Murmurs thus produced are almost invariably systolic in time. When the tracheal and bronchial glands are enlarged a peculiar purring sound may be audible on auscultation over the upper part of the sternum when the patient holds his head far back (Hare). The condition of the vocal resonance is variable; it is often absent over a growth, but bronchophony may be present.

Mensuration.—A cyrtometric tracing may prove that one side is retracted or that there is local bulging. General enlargement is less common than retraction, a fact which is no doubt due to the frequency of pulmonary collapse. A co-existing pleural effusion may give rise to enlargement of the affected side.

COURSE AND DURATION

The course of the disease is not uniformly progressive, as intermissions of some duration may occur, during which there is relief from the most severe symptoms. The growth of a scirrhous tumour is slow, whereas sarcoma and encephaloid cancer tend to develop more rapidly.

The extension of the growth is generally marked by gradually increasing dyspnœa, which, if not previously of that character, often becomes paroxysmal towards the close, and the patient may die during an attack of the kind. In other cases death is due to gradual exhaustion, or to cardiac failure from increasing dilatation of the right cavities of the heart, or to a secondary growth in the brain giving rise to paralysis or convulsions. Acute pericarditis not uncommonly precedes the fatal termination.

The average duration of the cases which we have analysed was ten months, the longest period being two years and the shortest ten weeks. A case is, however, on record in which death occurred nine days after the onset of dyspnœa, which was the first symptom observed. On the other hand, two are recorded in which life was prolonged for five and seven years respectively.

PROGNOSIS

The prognosis is necessarily fatal, but it may be well to recall the fact that a syphilitic gumma growing from behind the sternum may simulate a sarcoma of the anterior mediastinum, and that under appropriate treatment a tumour of this kind may disappear.

DIAGNOSIS

There are few conditions which may give rise to greater difficulties in diagnosis than that under consideration, and, owing to the absence of characteristic symptoms, cases of the kind under the care of the most competent physicians often pass unrecognised.

The prominence generally given in textbooks to the differential diagnosis of aneurysm and mediastinal tumour might lead to the impression that, as a rule, these are the two conditions most likely to be confused. Practical experience, however, proves that cases of tumour are far more often mistaken for examples of 'phthisis,' chronic pneumonia, bronchiectasis, emphysema, and bronchitis, or of pleural or pericardial effusion, than aneurysm.

Adenoma of the lymph glands is a form of tumour of very varying malignancy, and it may originate in the tracheal and bronchial glands. As already stated, it is classed by some writers with lymphosarcoma as 'malignant lymphoma'; but we hold that there is clinically a well-marked difference between a typical case of Hodgkin's disease involving the mediastinal glands and one of round-celled sarcoma of the mediastinum; and lymphadenoma may therefore be excluded in considering the question of differential diagnosis. Lymphadenoma is characterised by

1. The presence of great anæmia and dropsy.
2. General enlargement of the lymphatic glands throughout the body.
3. A growth in the spleen similar to that in the glands.

4. A febrile temperature.

5. Enlargement of the mediastinal glands occurring in association with a glandular enlargement elsewhere.

Pulmonary tuberculosis.—In cases of pulmonary disease the examination of the sputa for tubercle bacilli should be considered as much a matter of routine as that of the urine for albumen in disease generally. If there are destructive lesions in the lungs and there is copious expectoration, but after repeated examination of the sputa no bacilli are found, it is practically certain that the case is not one of tuberculosis.

Reliance cannot be placed upon such symptoms as hæmoptysis, emaciation, night sweats, and pyrexia, as they are common to both affections; although the degree of fever is, generally speaking, less in cases of tumour. The same is true with regard to such physical signs as are merely indicative of pulmonary conditions, which may be present in either case; but great importance attaches to the site of the lesions, and the absence of the 'line of march' peculiar to tuberculosis is often of great assistance in the differential diagnosis of the two conditions.

Chronic pneumonia, or fibrosis, is unaccompanied by signs of pressure, and the duration of the disease may have been much more prolonged than is usual in cases of tumour.

The physical signs favouring a diagnosis of tumour are absolute dulness on percussion, feebleness or absence of breath sounds, diminished vocal fremitus and resonance, and absence of adventitious sounds. Retraction may be present in either condition, but it is usually much more marked in chronic pneumonia. Limitation of the signs to one lobe, often the lower, points to a diagnosis of chronic pneumonia.

Pleurisy with effusion and empyema.—The discussion of this point might be almost indefinitely extended, but to do so can hardly be necessary, as in practice the question is in doubtful cases invariably decided by the results obtained on puncture with an aspirator needle, repeated, if necessary, at different sites. It is true that a tapping may be 'dry' although fluid is present, but perseverance is generally ultimately rewarded with success; and if the diagnosis cannot be cleared up in this way, it can rarely be determined by a nice consideration of the relative value of certain physical signs. The two affections are often associated, and the diagnosis may then present great difficulty. If paracentesis shows that a blood-stained effusion is present, and after it has been withdrawn the heart does not return to its normal position, the case is probably not one of uncomplicated pleurisy. The heart may, however, not return to the normal position immediately after paracentesis in a case of pleurisy when the effusion is not due to the presence of a growth. In a case recently under the care of the writer at the Brompton Hospital, after 30 ounces of clear serous effusion had been withdrawn from the left pleura there was no change in the position of the heart, which was markedly displaced to the right. This fact led to a suspicion that a growth was present, but three days after the tapping the patient stated that he had felt his heart 'give a jump' during the night, and on examination the heart was found to be in its normal position.

The fluid in cases of mediastinal tumour is, however, more often serous than hæmorrhagic (2 : 1 Moutard-Martin), and the effusion may be blood-stained, apart from the presence of either cancer or tubercle.

Careful examination after the fluid has been withdrawn may reveal the continued presence of dulness at the upper part of the chest, and if this should transgress the middle line the diagnosis of a growth would be almost certainly correct. But in some cases in which a growth is complicated by pleural effusion the fluid will not flow owing to the fact that the lung cannot expand. Microscopical examination of the fluid should not be neglected, but it is rarely attended with results which are conclusive.

Bronchitis and emphysema.—If not dependent on local causes both these affections are bilateral and symmetrical. Experience also furnishes us with a standard of the degree of dyspnœa which corresponds to lesions of a given extent, and when severe dyspnœa coexists with a slight amount of bronchitis or emphysema, the possibility that a growth may be present should be borne in mind. Hæmoptysis, which so often occurs in cases of tumour, is not a symptom of either of these affections.

Syphilitic stenosis of the trachea or main bronchi may give rise to symptoms and physical signs very closely simulating those of mediastinal tumour. All the usual symptoms of many chronic pulmonary diseases—such as cough with expectoration, dyspnœa, wasting, and hæmoptysis—are common to both, but signs of pressure upon the vessels and nerves are absent in cases of syphilitic stenosis of the trachea or main bronchi. The history may also give a clue to the diagnosis, as such cases are generally of prolonged duration; it may, however, in some cases be necessary to wait before giving an opinion. (The condition is fully considered in the chapter on Pulmonary Syphilis.)

Bronchiectasis.—It must be remembered that bronchiectasis is a secondary affection, and in every case of that disease an attempt should be made to determine the primary cause. In this way, by a process of exclusion, the possibility of the presence of a growth is brought to mind. When the two affections coexist and all distinctive signs of tumour are absent, the diagnosis presents great difficulty.

Aneurysm of the aorta.—In order to give it the prominence we believe it deserves, we place first the presence of *tracheal tugging*. This is never met with, so far as we are aware, in tumour; on the other hand, it is rarely absent in aneurysm when the sac is in contact with the trachea or left bronchus. The value of this sign has been doubted; but from the time of its discovery by Surgeon-Major Oliver[1] the writer has given careful attention to it, and the outcome of his experience is as above stated.

In aneurysm the evidence of venous obstruction is, as a rule, much less marked than in tumour, and consequently the appearances of the head, face, neck, and arms which characterise some cases of tumour are absent. An impulse of a heaving character may be present in either affection; but, even when communicated to a growth which has perforated the chest wall, the impulse is never expansile as in aneurysm.

In cases of aneurysm the pulsation is usually in proportion to the extent of the dulness, whereas in cases of mediastinal tumour an absolutely dull note may be found over a large area with only a moderate degree of pulsation.

A murmur may be produced by either condition, but in cases of tumour it lacks the peculiar 'whirling' character often noted in aneurysm and does not extend into the diastole.

The latter statement does not, however, imply acceptance of the view that a systolic and diastolic murmur of true aortic rhythm may occur in aneurysm apart from aortic regurgitation.

The presence of cardiac hypertrophy is usually given as a point in favour of aneurysm. Aneurysm of the aorta and hypertrophy of the heart may certainly be found in association, but in such cases both arise from a common antecedent cause or valvular disease is also present, and in neither case is the aneurysm the cause of the cardiac hypertrophy. The size of the heart is often unaffected in aneurysm, whilst the right cavities of the heart may be greatly enlarged in cases of tumour. Inequality and retardation of the radial pulses are conditions less commonly observed in cases of tumour than in aneurysm.

Laryngeal paralysis may occur in both affections, but is also less common in cases of tumour.

From pericardial effusion.—The diagnosis of a pericardial effusion from a growth involving the anterior mediastinum may be attended with considerable difficulty. The typical outline of the dull area in the former condition is pyramidal, whereas in the latter it is usually irregular, and most extensive beneath the manubrium. Elevation of the apex beat is common in pericardial effusion, but is rarely observed in cases of tumour, except when marked retraction of the chest wall is present. With a large effusion in the pericardium no impulse may be appreciable, and such a condition may certainly simulate a growth overlying the heart; but a tumour in such a position is rarely met with. The shifting of the margin of the dulness and of the site of the impulse with varying positions of the patient is diagnostic of pericardial effusion, but unfortunately it is a sign which cannot always be made out. The marked effects of pressure on the veins is absent in pericardial effusion, but a faintly cyanotic tint not uncommonly accompanies extreme degrees of that condition. Laryngeal paralysis has been observed by Baumler in a case of pericardial effusion; it disappeared with the absorption of the fluid. Such a condition must be extremely rare. In the differential diagnosis of these two conditions the mode of onset of the illness and the character of the pyrexia will necessarily influence the diagnosis.

Lesions of the pericardium are by no means uncommon in cases of mediastinal tumour, as is shown by the fact that in the thirty cases which we have analysed, acute pericarditis was present in four, in one of which the sac contained a pint of sero-fibrinous exudation, and in six cases the pericardium was either invaded by or adherent to the tumour.

The site of maximum intensity of the cardiac sounds has been observed to be at a point distant from the heart in cases in which both tumour and pericardial effusion were present.

Cancer of the œsophagus may extend to the mediastinal glands, and the case in its later stages may thus practically become one of tumour in that region. There may be little in the history of the case to suggest that the œsophagus was the primary seat of the disease, as in some cases the new growth undergoes ulceration and softening almost as quickly as it forms, and thus it may never cause a sufficient degree of obstruction to give rise to marked difficulty in swallowing.

Laryngeal paralysis.—The discovery of paralysis of the abductor muscles of one vocal cord may be the first event to arouse a suspicion of the presence of an intrathoracic aneurysm or tumour. In such a case it must, however, be borne in mind that the fixation of the cord may be due to some local disease which has led to ankylosis of the crico-arytænoid joint, or to a central or peripheral lesion of the pneumogastric or recurrent laryngeal nerve. On the right side it may result from thickening of the pleura over the apex of the lung.

Paralysis of the abductors has also been met with in various chronic pulmonary affections—*e.g.* chronic pneumonia and anthracosis—and it may follow calcification of the peritracheal and bronchial glands.

As already stated, abductor paralysis from pressure upon the left recurrent laryngeal nerve is more common in cases of aneurysm than of tumour, and bilateral abductor paralysis is not often met with as a result of intrathoracic disease.

Diagnosis of the nature of the growth (sarcoma and cancer).—The presence of a large growth, as shown by well-marked physical signs and pressure effects, points strongly to sarcoma. A growth primarily situated in the anterior mediastinum, or extending to that portion of the space, is also probably a sarcoma, as cancerous growths far more frequently originate in the posterior mediastinum. This statement, however, does not imply that the majority of tumours in the posterior mediastinum are cancerous, as they are almost certainly sarcomata.

A growth which softens and breaks down or ulcerates into neighbouring structures or induces gangrene of the lung is more likely to be a cancer than a sarcoma. A tumour connected with the œsophagus is almost certainly a cancer.

Secondary deposits may occur with either variety of growth, but are more common in cases of cancer than sarcoma.

TREATMENT

In cases of substernal syphilitic nodes the administration of iodide of potassium in large doses and perchloride of mercury may give great relief; and a similar result may follow in some cases when there is no good reason for suspecting syphilis. As well-authenticated cases are recorded in which sarcomata of the skin have disappeared under the administration of arsenic, a trial may be given to that drug. Such an event, however, has not, so far as we know, ever been observed in a case of mediastinal tumour. If fluid is present in the pleura, its withdrawal by paracentesis may give temporary relief to the dyspnœa, and, if so, the operation may be repeated as often as the effusion reaccumulates, which as a rule it does in a comparatively short time.

Tracheotomy may possibly be required for the relief of urgent dyspnœa due to spasm of the larynx or bilateral paralysis of the abductors of the vocal cords, but such conditions are rarely present in cases of mediastinal tumour. The operation is, of course, useless if the trachea is directly compressed, either within the thorax or low down in the neck. The differential diagnosis of these conditions is considered on page 82.

Intubation of the trachea has been practised and has afforded temporary relief in cases in which the lumen of the trachea was narrowed by the compression of a post-sternal enlargement of the thyroid gland. Paroxysmal attacks of dyspnœa in cases of mediastinal tumour may be relieved by the injection of morphia and by the inhalation of oxygen. Stramonium and other remedies of the kind may also be of service for the relief of this symptom. A combination of ether with ammonia is generally useful as a stimulant to the heart and as an aid to expectoration.

J. K. F.

CHAPTER LXIV

DERMOID CYSTS

PAGE
Symptoms and diagnosis 695
Morbid anatomy 695
Case of intrathoracic tumour of a doubtful nature . . . 698

AMONGST the rare intrathoracic tumours mention must be made of dermoid cysts. A considerable number of cases have been described since the early part of this century, most of which have been in the mediastinum, but others have occupied one or other of the lungs, though in these cases it is more than likely that the tumour may have originally been developed in the mediastinum.

Symptoms and diagnosis.—When small they may cause no trouble; but as they enlarge—probably during adolescence or early adult life—they may give rise to symptoms or physical signs such as might be caused by any other simple tumours in the mediastinum or lung, and they might easily be confused with hydatid cysts or enlarged mediastinal glands. But usually in the course of time some complication arises, either as a result of the pressure of the mass upon important structures, or more probably from its rupture into the pleura or a bronchus, or the establishment of a communication between it and some large vessel. Thus pleurisy or empyema is liable to occur, and the expectoration of matter mixed with the contents of the cyst. In several of the recorded cases the patient noticed the presence of hairs in the expectoration. There may also be hæmoptysis. If the cyst communicate with an artery an expansile pulsation may be transmitted to it. It will not be forgotten that, like every other mediastinal tumour, a heaving pulsation may be transmitted to it by the heart or the large vessels. The prolonged expectoration of the contents of the cyst may also lead to the development of bronchiectasis or pulmonary tuberculosis.

Morbid anatomy.—The contents of these cysts resemble those of dermoid cysts elsewhere, but it seems that they share with ovarian dermoids the peculiarity of occasionally containing large fleshy processes covered with thick skin from which hairs grow. Figs. 150, 151 show a number of these processes removed by me from a case that is fully reported in the 'Medico-Chirurgical Transactions,' vol. lxii. 1890, p. 317. The patient, who was 29 years old, had had no symptoms, except slight shortness of breath, until she was seized with a sudden acute pain on the right side of the chest, six years before I saw her. This was the onset of pleurisy with effusion, which was followed by empyema. The empyema ruptured into a bronchus some years afterwards, and the patient on one or two occasions noticed a hair in the sputum and thought nothing of it. I recommended the opening of the empyema, and opened through the sixth right interspace, just outside the nipple line, a cavity three inches from the

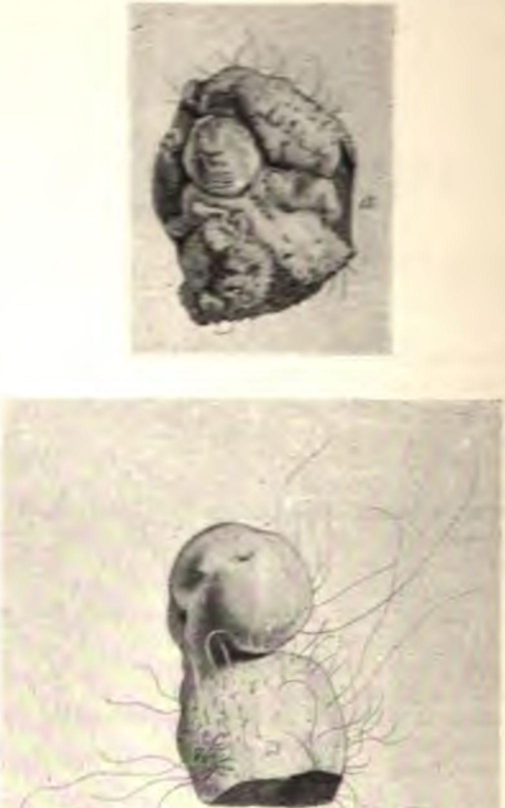


FIG. 150.—SHOWING FLESHY PROCESSES COVERED WITH THICK SKIN FROM WHICH HAIRS ARE GROWING

From a dermoid cyst of the lung.

surface, containing about an ounce of pus and packed full—as was discovered at a second operation—of a great felted mass of hair and fatty material which would have filled the palms of the two hands. Springing from the wall of the cyst—the deep part of which was lined by thick pink skin—were the fleshy masses shown in the figure. I removed them in order to try to make the cavity as simple as possible on a subsequent occasion, on which I also made the external opening very free. It was obviously impossible to attack the cyst wall itself; for it was evidently a large area of skin practically growing on the outer surface of the pericardium.

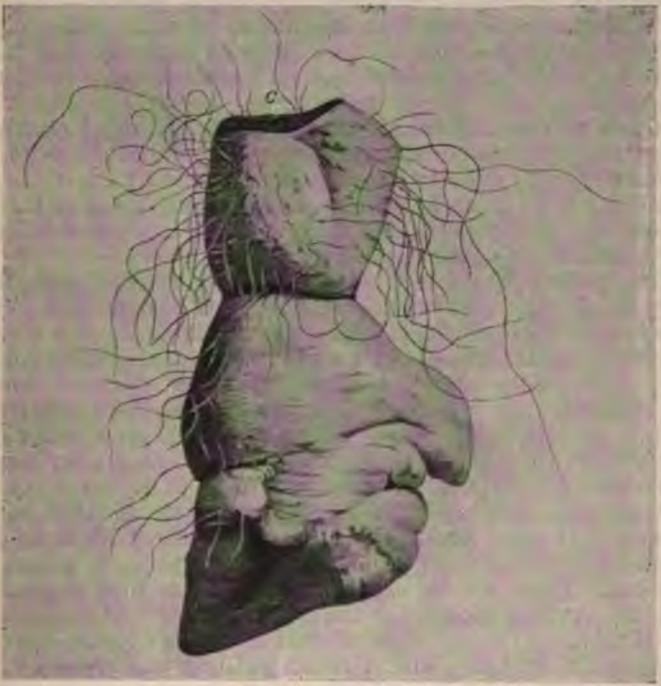


Fig. 151.—Showing fleshy Processes covered with thick Skin from which Hairs are growing

From a dermoid cyst of the lung.

The operation was, as far as it went, successful; that is, the patient was freed from the dangers attendant on the presence of accumulations of pus, and lost to a great extent her expectoration; but the cavity never dried as I hoped it would do, partly perhaps because of the constant escape of mucus into it from the bronchus. It therefore required daily cleansing, and, in spite of this, the discharge was never quite sweet. The patient, however, enjoyed fair health until some years later, when she died of some septic disorder.

A very similar case is figured in Alber's 'Atlas of Pathological Anatomy' (1833), and described by him: the patient died of phthisis.

A specimen of a dermoid cyst starting in the mediastinum and projecting into the right lung until it almost reached the surface and set up an empyema, was described at the Pathological Society this year, 1897, by Mr. Cyril Ogle. This patient suffered from severe hæmoptysis. The cyst contained a quantity of sebaceous material and a tooth. Springing from its interior were a number of the fleshy processes described above. The cyst communicated freely with one of the large bronchi, and its limits were very difficult to determine.

Case of intrathoracic tumour of doubtful nature. I have met with another strange case, which I should like to put on record here in order to give it greater publicity than it has yet obtained. It seems to fit in most easily amongst dermoid cysts, though why, if it were of this nature, it should have recovered after the treatment which was applied, remains a mystery.

The patient was a man aged 20, who suffered slightly from a dry cough from January to Easter 1891. Then one night, while he was away for his holiday, after a day's walking on the hills at Hastings, he felt languid, and went to bed at 11, and was awoke at 2 A.M. with sudden pain in the left side of his chest and behind the sternum, unaccompanied by sickness or faintness. Next day pleurisy was diagnosed. A week later a quart of clear yellow fluid was withdrawn from the left pleura, and a week after that a tumblerful of turbid fluid with flakes floating in it was drawn off. Between that time and October, when he was admitted to the Brompton Hospital under the care of Dr. Percy Kidd, he had suffered from some dyspnœa and a dragging pain on raising the left arm. There was no expectoration and but little cough. In August a swelling appeared at the epigastrium.

On admission he had signs of fluid in the left pleura, with displacement of the heart, and a round swelling was present at the epigastrium, to which the pulsations of the heart were transmitted.

On October 15, sixty-five ounces of dark blood-stained fluid were withdrawn from the left pleura, in which nothing suggesting a new growth was found microscopically.

The dulness was much diminished, but the swelling at the epigastrium was unaltered, and a patch of dulness remained behind the sternum, extending up to the first right cartilage.

On November 28 an incision was made into the epigastric swelling, which was becoming more tense, and a cavity was opened containing a considerable quantity (six or seven ounces) of brown purulent fluid, something like that previously extracted from the side, floating in which were numerous small rounded masses of a brilliant chrome-yellow pultaceous material, looking like the yellow palm oil used for lubricating the wheels of railway carriages. The cavity extended upwards behind the sternum, and on a subsequent occasion I endeavoured to discover its upper limit by making a hole through the middle of the sternum. The finger, however, was found to be amongst the large vessels, in the superior mediastinum, and I did not venture to go any further. An elaborate analysis of this fatty material was made by Dr. Halliburton, but it threw no light on the case. We never found any of the usual contents of dermoid cysts, and I do not therefore know whether the case should be placed here; but I am at a loss for any other explanation of it. The yellow material continued to escape for a considerable time, and ultimately the sinus closed. It remained healed for some time, but subsequently a small ulcer formed at the top of the sternum, which, after discharging for some months, finally healed, and he is now, or was in February 1897, in good health. During his stay in the hospital his temperature remained almost normal.

R. J. G.

CHAPTER LXV

CHANGES IN JOINTS AND BONES ASSOCIATED WITH INTRATHORACIC DISEASES

('PULMONARY OSTEO-ARTHROPATHY')

PAGE
Symptoms 701
Pathology 702
Diagnosis 705
Treatment 705

Amongst the complications of various chronic chest diseases are certain conditions of the joints and bones, to which considerable attention has been devoted during the last seven years. The chest diseases in connection with which these changes have been noticed are numerous, but are always chronic and usually suppurative; they are sometimes, but by no means always, tubercular. Amongst them may be mentioned empyema, bronchiectasis, pulmonary tuberculosis, and abscess of the lung. There is little doubt that at least two distinct diseases of joints, or bones and joints, have been included by different writers under the name which was coined by Marie in 1890—'Ostéo-arthropathie hypertrophiante pneumique.' I believe it will also be shown that they are not necessarily associated with disease in the thorax.

Great care is usually taken to distinguish the conditions which will be described below from acromegaly, a disease which appears to me to be quite distinct from them, and, besides its own easily distinguishable characters, to possess the peculiarity of being always associated with some disease of the thyroid gland, and generally, if not always, with some disease of the pituitary body. The reason for dwelling upon this point arises from the fact that, though to Marie undoubtedly belongs the credit of first drawing attention to the occasional association of thoracic trouble with bone and joint disease, the case which he originally brought forward was really one of acromegaly. This was one of the two celebrated brothers Hagner, who have in turn served to illustrate more than one form of disease. One of them has, however, died since he had become recognised as the type of pulmonary osteo-arthropathy; and a most exhaustive *post-mortem* examination by Julius Arnold has definitely shown that he was really the subject of acromegaly.

Having said so much it will be well to describe the characters which all the cases possess in common, and then to endeavour to distinguish between the two conditions which I believe go to make up the group.

Symptoms.—All the cases which have been described possess, in greater or less degree, clubbing of the fingers and toes, and attempts have been made to show that this modification in shape of the terminal digits differs from the ordinary clubbing (digiti Hippocratici). I believe, however, that such attempts have failed, and that the swelling is nothing more than the clubbing we are accustomed to see, independently of changes in joints and bones, but which, superadded to these changes, makes them wear an unusual aspect. The fingers are much swollen, and have been said to resemble in shape the legs of windsor chairs. Their joints are certainly swollen, and an examination of them almost always suggests more or less enlargement of the bones; but while it has been proved that the bones are sometimes enlarged, it is equally true that in some of the recorded cases there was no osteal enlargement at all. It has been said that there is no thickening of the soft parts, but it is certainly present in some cases.

A similar enlargement is present over the wrists and ankles; but the same remarks that were made about the bones of the hands apply to those of the legs and forearms.

Changes of the same kind, but usually less marked, have been observed in the elbows, knees, and hips.

There is a good deal of stiffness and some impairment of movement of the affected parts. The hands and the feet are cold and clammy, and perspiration from them is often excessive. Occasionally it is so great from the feet that the sheet is obviously wet if they are kept for any length of time in one position.

Amongst the accompanying phenomena in other parts of the body which have been recorded are the following: (1) The nose has been described as large and red at the tip—probably the clubbing of the nose to which frequent allusion has been made in this work. (2) Some modification of the shape of the upper jaw was described by Marie, but not by other observers. It may be suggested that this observation was made on the patient Hagner, who, as has been stated, was merely the subject of acromegaly. (3) Kyphosis has often been described. In some of the cases this was undoubtedly due to caries of the spine. It may sometimes have been merely the kyphosis which, as was pointed out on page 14, not infrequently results from prolonged dorsal decumbency. It is said to have principally affected the lower dorsal and lumbar regions of the spine. It must not be forgotten that many of the conditions in the chest in association with which these joint and bone changes have been met with, are liable to produce of themselves more or less distortion of the spine. Such, for example, is notoriously the case with empyema. (4) Retrosternal dulness has also been mentioned, but it is obviously unwise to make very much of this symptom, seeing how difficult it is to recognise, and how likely it is that, if it were present, it might be essentially connected with the chest trouble, and not with the joint or bone disease.

Pathology.—I believe that a large number of the cases, the clinical characters of which correspond with those given above, have no enlargement of the bones whatever. This conclusion is not, unfortunately, founded on any *post-mortem* observations, but only on the following facts:

First.—In one, at least, an examination by means of the Röntgen rays shows that the bones are apparently normal, and a careful examination of several patients with this affection has convinced me that such is often the case.

Secondly.—Two of the cases which have come under my observation, both of empyema, recovered completely within a short time of the cure of the chest condition. It seems scarcely conceivable that such would have occurred if the bone changes to be presently described had been present.

Both of these were described in a lecture delivered at the Brompton Hospital and published in the 'British Medical Journal,' July 11 and 18, 1896. The second (Case VI. in the lecture) was at the time a marked example of the disease. He subsequently expectorated his empyema, and within a few weeks his limbs had assumed an almost completely natural appearance.

These cases, therefore, though there is at present no evidence forthcoming as to the changes in the joints, appear to fall into the class of septic inflammations of joints at present included under the vague title of osteo-arthritis; but which, it may be hoped, will before long be definitely disassociated from that disease. They present a peculiar appearance because of the superadded clubbing of the fingers and toes, but are presumably closely allied to, if not identical with the cases of so-called osteo-arthritis occurring in young people, especially females, which are frequently associated, as was pointed out by Dr. Ord, with chronic leucorrhœal or other discharges.

In another class of cases, such as those described by Bamberger, Thorburn, and others, marked changes in the bones and joints have been observed during life, and demonstrated in a few instances by *post-mortem* examination. The changes in the bones consist of a more or less extensive sub-periosteal deposit of bone. In some it has affected the extremities of the long bones only, in others the whole length of the diaphyses has been involved in the change. The phalanges have been much thickened, and there have sometimes been found erosions of the articular cartilages. Some of these patients have been undoubtedly the subjects of tuberculosis, and it has been suggested by Thorburn that the bone and joint changes are essentially tubercular. This is a very fair suggestion, and it is important to notice that in the last case carefully recorded by him in the 'Transactions of the Pathological Society of London,' vol. xlvii., 1896, p. 177, 'the chest symptoms were never prominent,'—a most suggestive observation. One wonders if the chest trouble had anything to do with it at all.

It is also to be noted that in few, if any, of the cases is any mention made of a careful inquiry into the possibility of a syphilitic taint having been present, especially as the changes described in the articular cartilage seem to have been very similar to those observed in some cases of syphilitic arthritis.

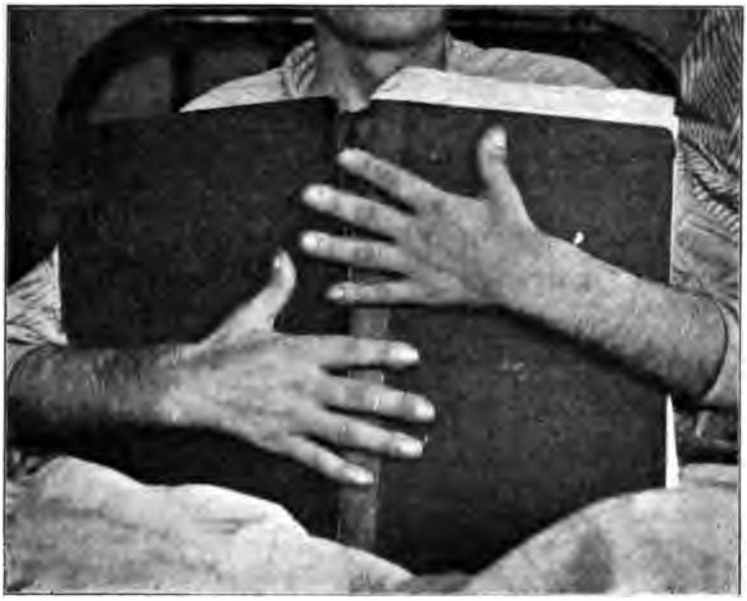


FIG. 152.—HANDS OF PATIENT IN WHOM THERE IS OBVIOUS BONY ENLARGEMENT

Under the circumstances it does not seem wise to give any elaborate illustrations of the affection of the bones, but the reader is referred for them to Thorburn's paper mentioned above, and to one by Bamberger ('Zeitschrift für Klinische Medecin,' 1891, p. 193).

It may however be interesting to compare the accompanying photographs of two cases, in one of which (fig. 152) there is undoubtedly thickening of bone as felt by the finger and shown in the skiagram (fig. 153), while in the other (fig. 154) there was no clear evidence of any bony enlargement.

The patient from whom fig. 154 was taken is reported in the lecture mentioned above. He was twenty-five years old, and was the subject of chronic bronchiectasis. The joint troubles had started six months before admission.

The patient from whom fig. 152 was taken is a man forty-one

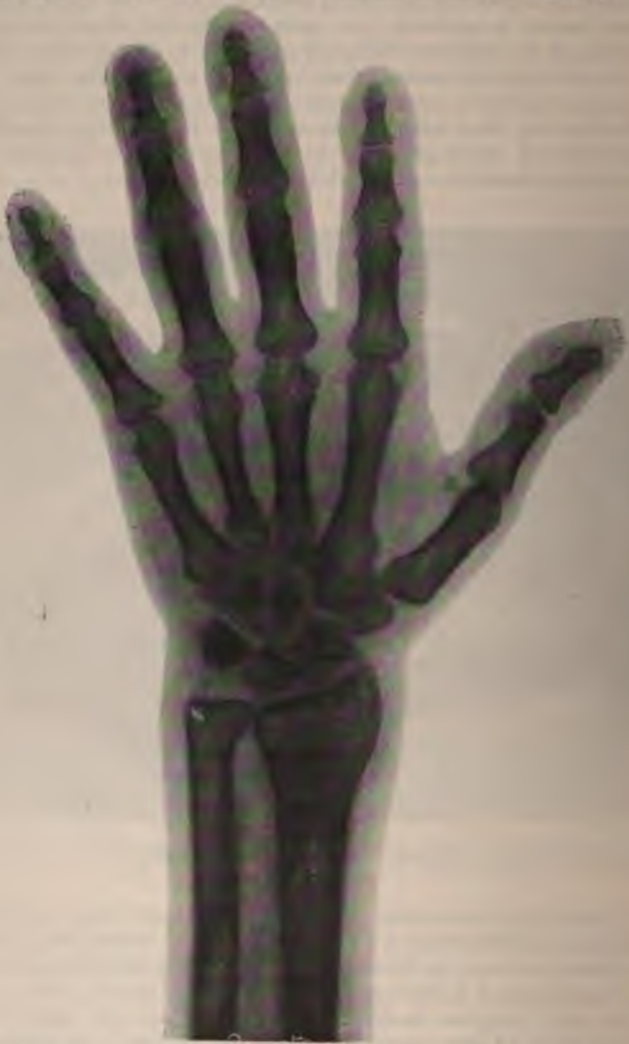


FIG. 153.—SKIAGRAM OF HANDS AND FOREARM OF PATIENT IN WHOM THERE IS OBVIOUS BONY ENLARGEMENT

years of age, recently in University College Hospital. He was the subject of a large cavity in the lower lobe of the right lung, which might well be tubercular, though no tubercle bacilli were ever found either in the pus or in the sputa. His chest troubles were of almost two years' duration. He had had so-called rheumatic pains for twelve years, but the swelling of hands and arms dated only from about a year back.

Diagnosis.—After having suggested a doubt as to whether the disease under discussion is a distinct pathological entity, it would be inconsistent to lay down definite rules by which it may be distinguished from other morbid states. It must suffice to mention those with which the conditions which have been described are likely to be confused. These are osteitis deformans, acromegaly, tubercular or syphilitic bone and joint lesions, and osteo-arthritis. Osteitis deformans and acromegaly have such clearly defined characteristics that they ought not to lead to any further confusion, even although they must occasionally be associated with intrathoracic disease. From the other three diseases it is impossible to distinguish many cases of 'pulmonary osteo-arthropathy' if, as has been hinted, they are often identical with one or the other of them.

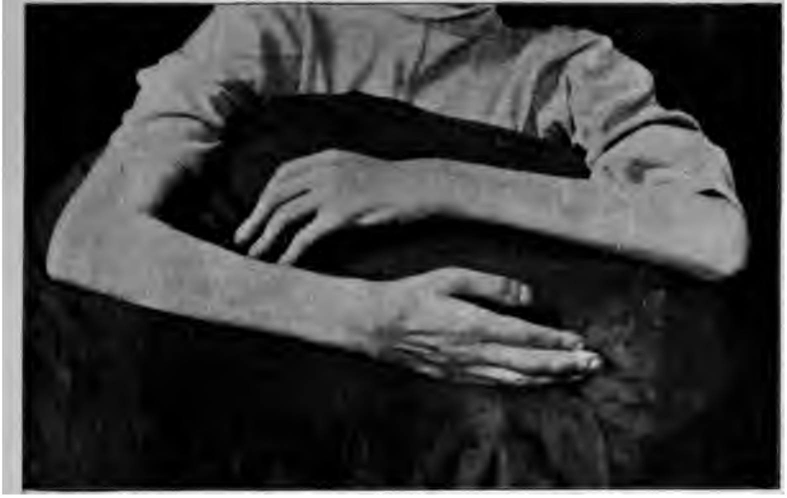


FIG. 154.—HANDS OF PATIENT IN WHOM THERE WAS NO EVIDENCE OF BONY ENLARGEMENT

Treatment and prognosis.—Under this head there is nothing to be said, except that the cases in which there is no thickening of the bones will almost certainly recover if the accompanying disease in the chest is cured. None of the recorded cases appear to have been very painful, and no special treatment has been required or used for the osteo-arthropathy itself.

CHAPTER LXVI

CLUBBING OF THE FINGERS AND TOES

THIS condition is present in so many forms of pulmonary disease that some account of it appears to be necessary.

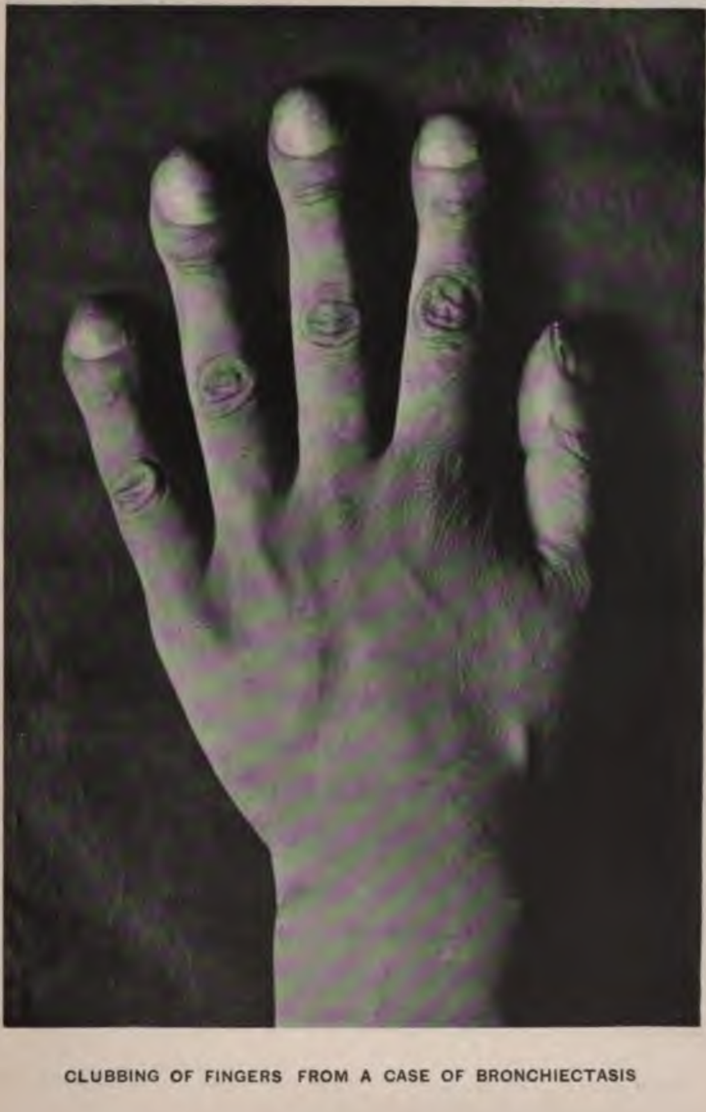
A very interesting paper on the subject by Dr. Samuel West will be found in vol. xxx of the 'Transactions of the Clinical Society,' and to this source the writer desires to acknowledge his indebtedness for some of the facts and references here given.

The affection was known to the ancients, and was in later times referred to as the 'Hippocratic deformity of the fingers.' The following description of it is contained in Trousseau's 'Clinical Lectures' (iii. 305, Syd. Soc.):—

'This deformity consists in contraction of the ungual phalanx, with enlargement and thickening of the digital pulp. Whilst the nail becomes curved towards the palm of the hand, the extremity of the finger assumes the form of the large end of a club, and sometimes in enlarging it flattens so as to resemble the head of a serpent. This deformity generally comes on by slow degrees, but at other times it is produced with great rapidity, the patient suffering pain from the change which is going on. In some persons the toes are the seat of a similar deformity; but when it occurs in the toes it is generally in a much less degree than in the fingers.'

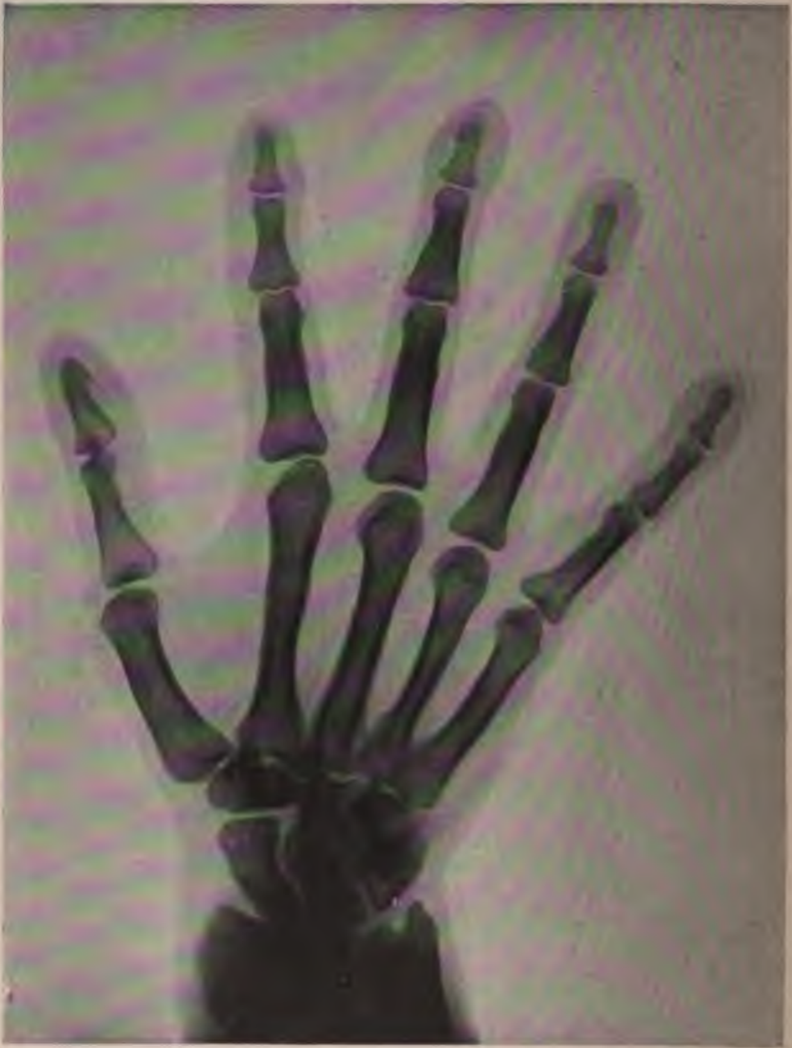
We may mention that it sometimes affects the nose also. Little need be added to Trousseau's description, except a fact which we do not remember to have seen stated—viz. that when true clubbing of the fingers is present, if the observer places one of his fingers upon the upper edge of the nail at its free margin, *i.e.* the distal end, and his thumb upon the under surface of the clubbed finger, and presses downwards, the hard margin of the root of the nail can be distinctly felt if another finger is applied over it. We have never noticed this in a case where clubbing was absent. It shows that the nail is raised from its bed, possibly by an overgrowth of the subjacent papillæ.

The bulbous ends of the fingers are in some cases cyanotic, and



CLUBBING OF FINGERS FROM A CASE OF BRONCHIECTASIS

PLATE V.



SKIAGRAM OF THE SAME HAND, SHOWING ABSENCE OF ENLARGEMENT OF THE BONES the skin over the root of the nail is invariably shiny and presents a tense appearance.

The exact nature of the changes present are not known. In a specimen which we removed for microscopical examination the condition entirely disappeared, as a result of preservation in spirit. It has been ascribed by Buhl[1] (quoted by West) to a fibrous thickening of the rete mucosum. It is not due to chronic venous congestion, as it may occur apart from any affection in which such a condition is present. Clubbing of the fingers accompanying a general, as distinguished from a local, affection is bilateral and symmetrical.

It may, however, be unilateral, as in a case of subclavian aneurysm, in which it was only present on the same side of the body as the aneurysm.

With whatever affection it is associated, it may disappear entirely with the disappearance of the disease.

Etiology.—Clubbing may arise without obvious cause in persons apparently in good health, and may persist or disappear. It sometimes attains to a considerable degree in quite a brief period. West mentions a case in which he observed this to occur in less than a fortnight. In a case of chronic non-tubercular pulmonary disease, which the writer thought to be due to the rupture of a bronchial gland into a bronchus, it attained to a considerable degree in less than two months. The diseases in which it is most marked are bronchiectasis, in which affection it is so common as to be of some slight value as an aid to diagnosis and in such cases is not uncommonly associated with clubbing of the nose, and also in chronic pulmonary tuberculosis with excavation, emphysema with chronic bronchitis and bronchial asthma, empyema, and congenital morbus cordis.

It has also been observed in a variety of other affections, such as purpura, pneumonia, and pulmonary abscess. Trousseau states that he observed it in cases of 'nervous asthma.'

J. K. F.

---

[1] *Lungen-Entzündung*, 1872, p. 159.

INDEX

An indexed Table of Contents will be found at the head of each chapter, which may be referred to for subjects omitted from the General Index.

ABSCESS OF LUNG (general), chap. xix. 241 ff.
  surgery of, 417 ff.
Acromegaly, 700, 701
Actinomyces, 456
ACTINOMYCOSIS (general), chap. xxxviii. 453 ff.
Aegophony, 66, 561
Alar thorax, 52
Amphoric breathing, 60
    echo, 64
Anaesthesia in operations on the chest, 418
Aneurysm, compression of bronchus by, 151
  compression of trachea by, 82
  perforation of trachea by, 83, 84
  pulmonary, chap. xlvi. 514 ff.
Anthracosis, chap. xxiii. 269
Apex right, variations in breathing, 373
Apoplexy pulmonary, 503 ff.
Aspergillus, 460 ff.
ASTHMA (general), chap. xiv. 183 ff.
  over-distension of lung in (mechanism of), 161, 188
  bronchial, 170, 183, 106
  cardiac, 183
  hay, 183, 188
  renal, 183
  aura in, 184
  compressed air bath in, 192
  nasal disease and, 186, 192, 194
  emphysema in, 189
Atelectasis, 285, 291
Atheroma of pulmonary vessels, 512 ff.
Atrophy of lungs, senile, 166
Auscultation, 58 ff.
Auscultatory percussion, uses of, 55

BACELLI's sign, 65, 562
'Barrel-shaped' chest, 52
Basic pulmonary lesions, 357
Bell sound, 64, 627
  over cavity, 639
  in subdiaphragmatic abscess, 618
Bones, affection of, in pulmonary disease, 700 ff.
Breath sounds, varieties of, 59 ff.
Bronchi, scheme of, 42 ff.
Bronchial arteries, 48
  asthma, 170, 183
  asthma in tuberculosis, 344
  breathing, 59
  casts, 120
  veins, 48
BRONCHIAL STENOSIS AND OBSTRUCTION (general), chap. x. 150 ff.
  results of, 129
BRONCHIECTASIS (general), chap. viii. 123 ff.
  acute, 131, 134
  causation of, 128 ff.
  pleurogenous origin, discussion of, 130
  in stenosis, 129
  stages of, 134
  sputa, characters of, 132, 133
  haemoptysis in, 133
  pulmonary aneurysm in, 515
  clubbing in, 133
  amyloid disease in, 128, 133
  septic complications, 134
  cerebral abscess, 128, 137
  creasote vapour baths, 138 ff.
  intra-tracheal injections, 139, 141
  and tuberculosis, 357, 360
  in mediastinal growth, 683, 684
  surgical treatment of, 421 ff.
BRONCHIECTASIS IN CHILDREN (general), chap. ix. 143 ff.
Bronchiolectasis, 143
BRONCHITIS, ACUTE (general), chap. iv. 87 ff.
  liability to, in spinal curvature, 90
    in valvular disease, 90
    in arrested tuberculosis, 107
    in the tuberculous, 117
  sputa, a guide to prognosis in, 91
  hypostatic pneumonia in, 92
  tuberculosis, diagnosis from, 93, 96
  bronchial stenosis, diagnosis from, 107
  opium in (rules for use), 98, 99
  emetics in, 101
  oxygen in, 101
  venesection in, 99

BRONCHITIS, CHRONIC (general), chap. v. 103 ff.
BRONCHITIS, PLASTIC (general), chap. vii. 119 ff.
Bronchitis, capillary, 94
    gouty, 116
    putrid, 114
    syphilitic, 117, 434
Bronchophony, 65
BRONCHO-PNEUMONIA (general), chap. xxi. 252 ff.
    micro-organisms in, 252
    diagnosis from tuberculosis, 256
    and syphilis, 438
Bronchorrhœa, 113
Bruising of the lung, 494
Bruit d'airain, 64, 627
Bruit de pot fêlé, 57

CAPILLARY bronchitis, 94
Cardiac disease, liability to bronchitis in, 90; loss of murmur, 95
Cardio-pulmonary murmurs, causation of, 67 ff.
    diagnosis of, 69, 70
Casts, bronchial, 120
Catarrh (dry) of Laennec, 113
Cavernous breathing, 49
CAVITIES, PULMONARY, SURGICAL TREATMENT OF (general), chap. xxxvi. 416 ff.
    operation, steps of, 418 ff.
Cerebral abscess in bronchiectasis, 128, 137
    in empyema, 565, 616, 617
Charcot-Leyden crystals, 186
Chest, variations in shape, 32
Chloroform, preference for, 419
CHYLOTHORAX (general), chap. lx. [illegible] ff.
    normal conditions in repeated cases, 656, 657
    analysis of fluid, 652
Cirrhosis of lung, chap. [illegible] 288 ff.
    pleurogenous, 287
    [illegible], 288
    and syphilis, 439
[illegible] (general), chap. [illegible] 705 ff.
    pathology of, 706
    diseases causing, 706
    in emphysema, 171
    in new growths, 462, 466
    in [illegible], 469 ff.
[illegible] of lung, [illegible]
Cogwheel breathing, 50
Collapse of lung (general), chap. xxv. 290 ff.
    from muscular paralysis, 296
    diagnosis from consolidation, 292
    a cause of bronchiectasis, 125
Commotio thoracis, 499
Compressed air bath, 112, 178
    theory of action, 178
CONGESTION OF LUNG (general), chap. xxiv. 276 ff.
    acute, etiology of, 278
    chronic, chap. xxv. 282 ff.
Consolidation, diagnosis from effusion, 226 ff.
Contusio thoracis, 499
Cough and emphysema, 166
    (spasmodic), due to pressure, 157
Creasote vapour baths, 138 ff.
"Crepitation," 64
Curschmann's spirals, 187
Cyanosis in emphysema, 170
Cyrtometry, 46

DERMOID CYSTS (mediastinal and pulmonary) (general), chap. lix. 686 ff.
    hairs in expectoration, 686
DIAPHRAGM, DISEASES OF (general), chap. lxi. 693 ff.
    height of, 9, 10
    wounds of, 338, 340
Diaphragmatic hernia, 338-342
    pleurisy, 670
    paralysis (collapse of lung in), 696, 698, 296 ff.
    (sub-) abscess, 617, 619
Diphtheria
    paralysis of diaphragm in (effect on lungs), 699 ff., 296 ff.
    of trachea, 73
Displacement murmurs, 67 ff.
Dulness, definition of, 56
    significance of, 57
DUST, INHALATION OF, DISEASES DUE TO (general), chap. xxiii. 260 ff.
    tubercular origin of, 260, 270, 275
    colour of sputa in, 270, 272
Dyspnœa, paroxysmal, in tracheal stenosis, 77
    in aneurysm, 150
        mediastinal growth, 695, 696, 697
    treatment of, 82

ELASTIC TISSUE, significance of, 190
    detection of, 187, 188
Embolism of Pulmonary Artery, 500 ff.
Emetics, uses and dangers of, 101
EMPHYSEMA (general), chap. xii. 159 ff.
    hæmoptysis in, 162
    large-lunged, 165
    small-lunged, 166
    atheroma of pulmonary artery in, 166, 512
    compensatory (mode of production), 167
    and tuberculosis, 167, 168, 169, 170
    pneumothorax from, 627, 628
    anastomotic circulation in, 165
    shape of chest, 171
    spinal curvature in, 171
    compressed air bath in, 178
    surgical, 498
EMPHYSEMA, INTERSTITIAL (general), chap. xiii. 181 ff.
Empyema, 554 ff.
    bacteriology of, 556 ff.
    loculated, 554

- Empyema (*cont.*)
  - tuberculous, 551, 584, 585, 591
  - calcified, 554, 608
  - from spinal caries, 622
  - in new growth, 690
  - simulation of, by spinal abscess, 622, 623
  - simulation of, by costal abscess, 622, 623
  - deformity after, 607 ff.
  - spontaneous discharge of, 565, 615, 616
  - cerebral abscess in, 565, 617, 616
  - varieties of acc. to micro-organisms, 590, 591, 592
  - streptococcus, 591
  - tubercular, apyrexia in, 592
    - treatment, 592
  - pneumococcus
    - cure by aspiration, 591
  - with bronchial fistula, 614, 615, 616
  - SURGICAL TREATMENT OF (general), chap. liii. 593 ff.; chap. liv. 610 ff.
    - of localised, 613
    - diaphragmatic, 613
    - double, 614
  - resection of rib (steps of operation), 600 ff.
  - Estlander's operation, 610 ff.
  - method of closure after operation, 606
  - irrigation, 604
  - disappearance of clubbing, 609
  - disappearance of amyloid disease, 609
  - non-closure (causes of), 609, 614, 622
- Endothoracic fascia, 22
  - inflammation of, 621
- Enteric fever, bronchitis in, 115
- Eparterial bronchus, 44
- Estlander's operation (description), 610 ff.
- Excision of lung (partial), 424 ff.
  - for growth, 489 ff.
- Exocardial murmurs, 67 ff.
- Exploration of Pleura (method), 593, 594
  - of Lung (method), 594
    - hæmoptysis in, 46

- FEVERS, ACUTE, BRONCHITIS IN (general), chap. vi. 115 ff.
- 'Fibroid Phthisis' (Sir A. Clark), 320, 321
- Fibroid Tuberculosis, 342
- FIBROSIS PULMONARY (general), chap. xxii. 260 ff.
  - from pressure of growth, 265
    - aneurysm, 265
    - tuberculosis, 260
    - syphilis, 439
- Fistula in ano, 372
- Foreign body, in trachea, 85, 86
  - in bronchus, 425 ff.
    - pleura, 538
- Fremitus, varieties of, 53
  - vocal, variations in, 53
- Friction sounds (pleural), 61, 62

- GANGRENE OF LUNG (general), chap. xx. 244 ff.
  - in paralysis, 246
    - insanity, 246
    - pulmonary tuberculosis, 248
  - from embolism, 248, 361
    - fat embolism, 248
  - surgery of, 417 ff.
- Glanders, pulmonary lesions in, 534
- GLANDS, BRONCHIAL, DISEASES OF (general), chap. xi. 153 ff.
  - tuberculosis of, 154 ff.
  - and syphilis, 439, 440
  - perforation of trachea by, 83, 84
  - a cause of laryngeal paralysis, 693
- Gouty bronchitis, 116
- Guaiacol, hypodermic injection of, 139, 402, 251

- HÆMOPTYSIS (general), chap xlvii. 519 ff.
  - etiology, 519
  - from hyperæmia, 520
    - laryngeal vessels, 524
  - senile (arthritic), 524
  - spurious, 524
  - in emphysema, 170
    - tuberculosis, 521 ff., 382
    - hydatid, 475
    - bronchiectasis, 133
  - blood, characters of, 520
    - T. bacilli in, 523
  - differential diagnosis of, 523
  - absence of pulmonary signs, 524
  - subsequent pyrexia, 521
  - astringents in, 527
- HÆMOTHORAX (general), chap. lviii. 648 ff., 495, 538
  - excessive extent of physical signs in, 649
  - traumatic, 538
- Hay asthma, 183, 188
- Hernia of lung, 495 ff.
  - spontaneous, 496
  - traumatic, 496–8, 539, 540
  - diaphragmatic, traumatic, 539
    - congenital, 541
- 'Honeycomb' lung, 146
  - 'Path. Soc. Report' on a case of, 147
- HYDATID DISEASE OF LUNG (general), chap. xl. 472 ff.
  - hydropneumothorax in, 642
  - distinction from pleural effusion, 478, 481
  - danger of puncture, 482
  - surgical treatment of, 482 ff., 642
- HYDATIDS OF THE PLEURA (general), chap. xli. 480 ff.
- Hydatid of liver, 618
- HYDROTHORAX (general), chap. lvii. 645 ff.
  - analysis of fluid, 645, 6
  - method of treatment, 647
- Hyper-resonance, 57
- Hypertrophy of lung, 165, 174

- INDURATION, BROWN, OF LUNG (general), chap. xxv. 283

Infarction, hæmorrhagic, pathology of discussed, 503 ff.
Influenza, pulmonary complications of, 529 ff.
INJURIES OF THE LUNGS (general), chap. xliii. 493 ff.
Intercostal vessels, 16, 17
  nerves, 18
  neuralgia, 568
  spaces, width of, 7
  muscles, collapse of lung in paralysis of, 286 ff.
Internal mammary artery, 17
  in paracentesis pericardii, 17 *n.*
Intratracheal injections, 139, 141, 402
Irrigation of pleura, 596, 604, 643
  dangers of, 567, 8

JOINTS, affection of, in pulmonary disease, chap. lxv. 700 ff.

KOUMISS, 391

LACERATION of the lung, 495
Laryngeal paralysis, various causes of abductor, 693
Ludovici angulus, 8
Lungs, anatomy of, 28 ff., 163, 164
  interlobar fissures, lines of, 30
  shape of, in health, 30 ff.
  structures in root of, 36 ff.
  movements of, 40
  surface markings, figs. 98, 100
Lymphatics of lungs, 48
  pleura, 49
  thoracic wall, 18

MEASLES, bronchitis in, 115
Mediastinitis, 624, 625
  diagnostic sign of, 625
Mediastinum, anatomy of, 19 ff.
MEDIASTINUM, TUMOURS OF (general), chap. lxii. 675 ff.
  bronchiectasis in, 683, 684
  cough in, 685
  dysphagia in, 685
  dyspnœa, paroxysmal, in, 685, 686, 689
  hæmoptysis in, 685
  laryngeal affections in, 685, 686, 693
  murmurs in, 688
  ocular symptoms in, 680 ff.
  pressure symptoms in, 679 ff.
    on nerves,
      sympathetic, 680–2
      vagus, 686, 693
      phrenic, 686
      recurrent laryngeal, 686, 693
      herpes from, 685
    on trachea, 685, 686
      bronchi, effect on lung, 683, 684
      veins, 685
Mediastinal Tumours (*cont.*)
  pyrexia in, frequency of, 686
  pleuritic effusion in, 685, 690
  pericarditis in, 693
  sputa ('prune juice') in, 685
Metallic tinkling, 64
Murmurs, false, 67 ff.
MYCOSIS OF LUNG (general), chap. xxxix. 459 ff.
  aspergillus, 460 ff.
  oïdium, 468, 469
  streptothrix, 469

NASAL disease and asthma, 186, 192, 194
  obstruction and collapse (of lungs), 288
Nucleins, 400

ŒDEMA OF LUNG, chap. xxiv. 279
  acute, 281, 567
Oïdium and pulmonary disease, 468, 9
Opium, use of, in bronchitis, 98, 99
OSTEOARTHROPATHY, PULMONARY (general), chap. lxv. 700 *ff.*
  cure of, 702, 4
Oxygen, use of, in bronchitis, 101
  in broncho-pneumonia, 258
  in pneumonia, 232

PAIN IN DISEASES OF LUNG, chap. lxii. 672 ff.
Pain referred, areas of (Head), 673
Paracentesis thoracis, details, 595, 596
Paralysis, respiratory, effect on lungs, 286 ff.
Parasternal line, 51
Pectoriloquy, varieties of, 65
Percussion, methods of, 54, 55
  sounds, theory of, 55
    varieties of, normal, 56
      morbid, 57
Pericardial effusion, 569, 692
  purulent, 619
  complication of empyema by, 619
  surgery of, 619
Pericardium, paracentesis of, 17 *n.*, 620
  incision of, 619
Peripleuritis, 621, 622
  from spinal caries, 622
    caries of rib, 623
'Phthisis,' fibroid (Sir A. Clark), 320, 321
  pneumonic, 320, 336 ff.
  syphilitic, 440 ff.
'Phthisis ab hæmoptoë,' 365
PHYSICAL DIAGNOSIS (general), chap. ii. 50 ff.
Pleura, anatomy of, 21 ff.
  variations in limits of, 23 ff.
  relation to pericardium, 27
  physics of, 537, 629 ff.
PLEURA, DISEASES OF, SURGICAL TREATMENT (general) chap. liii. 593 ff., chap. liv. 610 ff

PLEURA, INJURIES OF (general), chap. xlix. 536 ff.
PLEURA, TUMOURS OF (general), chap. lx. 656 ff.
Pleural adhesions, a cause of laryngeal paralysis, 693
- friction, cardiac rhythm in, 62
- sounds, distinction from pulmonary, 62
- thickening and bronchiectasis, 127, 130

PLEURISY, ACUTE (general), chap. l. 543 ff.
- tuberculous origin of, 545–50, chap. lii. 580 ff.
- bacteriology of, 544 ff., 550 ff., chap. lii., appendix 590 ff.
- pneumococcus, 590, 544 ff., 550 ff.
  - simulation of pneumonia, 547, 590
  - absorption of purulent, 591
- streptococcus, 591
- staphylococcus, 592
- diaphragmatic, 670
- chylous, chap. lix. 650 ff.
- referred pain in, 568
- with effusion,
  - physical signs of, 559 ff.
  - upper level of dulness in, 560
  - positive pressure in, signs of, 562
  - analysis of, 553
  - passive, chap. lvii. 645 ff.
  - chylous, chap. lix. 650 ff.
  - in new growths, 690, 662
  - hæmorrhagic, 555, 664
  - pulsation of, 559
  - diagnosis of, serous from purulent, 571
    - from pericardial effusion, 569, 570
    - from consolidation, 226
  - paracentesis in,
    - indications for, 574
    - sudden death after, 567, 568
  - micro-organisms in, chap. lii. appendix, 590 ff.
- with purulent effusion, see Empyema

PLEURISY, CHRONIC (general), chap. li. 576 ff.
- tubercular, 584 ff.
- latent, 576
- clubbing in, 577
- with effusion,
  - treatment by drainage, 596
    - irrigation, 596

PLEURISY, TUBERCULAR (general), chap. lii. 580 ff.
- supervention of, on other diseases, 582 ff.
- chronic adhesive, 586
- pyrexia in, 589

Pleurodynia, 568
Pleuro-pericardial friction, 62
PNEUMONIA, ACUTE CROUPOUS, chaps. xv., xvi., xvii. 195 ff.
- abscess in, 222, 242, 243
  - operative treatment of, 417 ff.

Pneumonia (*cont.*)
- antitoxic serum in, 200, 234
- bacteriology of, 198 ff.
- blood in, 207, 208
- crisis, 212, 216, 217 (date of)
- complications, 208 ff.
  - influence of, chap. xvi. 224 ff.
- diarrhœa in, 221
- diagnosis, chap. xvii. 226 ff.
- empyema in, 224
- gangrene in, 222, 247
  - operation, 417
- hyperpyrexia in, 232
- immunity, 200
- ice-bag treatment, 231, 332
- intermittent, 223
- latent, 223
- lysis in, 217
- 'massive,' 205, 224 (signs of)
- morbid anatomy, 202 ff.
- nervous symptoms in, 215
- pleurisy, simulation by, 201
- posture in, 224, 228
- pyrexia, 217 (variations), 221, 223
- physical signs, 213 ff.
- pericarditis in, 224
- prognosis, 229
- sputa in, 211, 216
- treatment, chap. xvii. 230 ff.
- unresolved, 129, 130, 236
- variations (influence of), chap. xvi. 215 ff.
- venesection in, 233
- 'wandering,' 223

PNEUMONIA, CHRONIC (general), chap. xxii. 260 ff., 236
- SUBACUTE INDURATIVE (general), chap. xviii. 237 ff.

Pneumonia, interstitial, chap. xxii. 260
  - in congenital syphilis, 431
- hypostatic, chap. xxiv. 279
- serosa, 281

Pneumoconiosis, chap. xxiii. 269
Pneumomycosis, chap. xxxix. 459 ff.
Pneumonectomy, 424, 489
PNEUMOTHORAX (general), chap. lvi. 626 ff.
- composition of air in, 630
- absorption of, 630, 641, 642
- positive pressure in, 630, 638
  - treatment, 642
- physical signs, of air, 636, 637
  - air and fluid, 636, 637
- diagnosis from cavity, 639
- in apparent health, 628
- in emphysema, 627
  - interstitial emphysema, 181
- from spontaneous evolution of gas, 628, 644
- in pulmonary tuberculosis, 630 ff. (for details see under Tuberculosis)
- traumatic, 494, 627; (production and cure) 537
- surgical treatment of, 641 ff.
- hydropneumothorax, 642
- pyopneumothorax, surgical treatment of, 642 ff.

Pneumothorax, pyopneumothorax (*cont.*)
drainage in, 643
irrigation of, 643
'subdiaphragmatic,' 618
Pressure, mediastinal, symptoms of, 155, 156, 187, 679 ff.
on vagus, asthma from, 187
Protozoa and pulmonary disease, 469 ff.
Pseudo-tuberculosis, 379
PULMONARY ARTERY, ANEURYSMS OF (general), chap. xlvi. 514 ff.
in bronchiectasis, 183
rupture from erosion, 518
THROMBOSIS OF (general), chap. xlv. 508 ff.
EMBOLISM OF (general), chap. xliv. 500 ff.
aneurysm in, 515
fat embolism, 501
air embolism, 501
and infarction, 503 ff.
Pulmonary vessels,
atheroma of, 166, 512 ff.
vasomotor control of, 277
dilatation of, 518
anatomy of, 46
Putrid bronchitis, 114
Pyopneumothorax (see Pneumothorax)

RALES, production of, 62
varieties of, 62 ff.
Resonance, vocal, 65
variations in normal, 65
Respiration, movements of ribs in, 5
Rhonchi, 62
Ribs, articulations of, 4
movements of, 5
anomalies of, 6
prominence of, 6
ossification of, 6
Rib, caries of, 623
esection of, 597 ff.
choice of site in, 604
hæmorrhage in, 603

SEROUS membranes, tuberculosis of, 587 ff.
Sibilus, 62
Siderosis, chap. xxiii. 269
Silicosis, chap. xxiii. 269
Skodaic resonance, 57
SPECIFIC DISEASES, ACUTE, PULMONARY COMPLICATIONS OF (general), chap. xlviii. 529 ff.
Spine, caries of, simulation of empyema by abscess, 622
scoliosis after empyema, 607
movements of thoracic, 2
Splenisation, 279
Sputa, elastic tissue in, 250
detection of, 367, 8
'prune juice,' 488, 685
hairs in, 696
Sternum, 8
anomalies of, 8, 9
angle, 8
Sternum (*cont.*)
caries of, 623
course of abscess, 623
surgery of, 624
Streptothrix and pulmonary disease, 469
Subdiaphragmatic abscess, 617, 618
Succussion splash, 64
Suction, post-tussive, 64
Sympathetic nerve, effects of pressure on, 680 ff.
SYPHILIS, PULMONARY (general), chap. xxxvii. 429 ff. (congenital and acquired)
gummata, examples of, 436 ff.
bronchitis and, 434
broncho-pneumonia and, 438
interstitial pneumonia in, 431
fibrosis and, 439
bronchial glands and, 439, 440
bronchial stenosis in, 151
destructive changes in (cases), 440 ff., 451
'syphilitic phthisis' (existence of discussed), 448
site of lesions in, 450
distinction from tuberculosis, 448, 9
congenital, and tuberculosis, 432 ff.
of trachea, 72–76
stenosis from, 78 ff.

THORAX, changes in
effect of posture, 13, 14
age, 13
sex, 14
disease, 14, 16
Thrombosis of Pulmonary Artery, chap. xlv. 508 ff.
Thyroid Gland, compression of trachea by, 80
Trachea, anatomy of, 40 ff.
TRACHEA, DISEASES OF (general), chap. iii. 71 ff.
stenosis of, 77 ff.
diagnosis from laryngeal, 77
Tracheal tugging (value of), 691
Tracheotomy, stenosis due to, 79.
interstitial emphysema in, 181, 182
Tubercle, structure of, 299 ff.
caseation, 302, 303
fibrosis, 304
Tubercle, bacillus of, 296 ff., 401
absence from sputa, 368, 369, 377. 378, 379
method of staining, 298
phagocytosis and, 301, 302
Tuberculin, 397, 398, 399
TUBERCULOSIS, PULMONARY, chaps. xxvii. to xxxv. 295 ff.
age and, 314, 315
albuminuria in, 358, 363, 383
altitude and, 314
alcohol in, 391
amyloid disease in, 362, 363
aneurysms (pulmonary) in, chap. xlvi. 514 ff.

Tuberculosis (*cont.*)
  antiseptics in, 401 ff.
  antitoxin in, 400
  apyrexial extension, 325, 344
  arrest of, 379 ff.
  blood in, 370
  basic, 357
  bronchitis in, 316, 383
  cancer and, 317, 316
  cardiac disease and, 316, 363
  CASEOUS, chap. xxxi. 336 ff.
    pyrexia, typical of, 327 ff.
  cavities in, formation of, 348, 349
    signs, 375, 376
    surgical treatment of, 423 ff.
  complete excavation of lung, 639
  CHRONIC (FIBRO-CASEOUS), chaps. xxxiii. & xxxiv. 346 ff.
  CLASSIFICATION OF, chap. xxviii. 318 ff.
  climatic treatment of, 408 ff.
  complications of, 358–363
    effect on prognosis, 382 ff.
  conditions predisposing, 313, 314
  congenital, 309 ff., 432 ff.
  cough, treatment of, 405, 406
  diabetes and, 316
  diarrhœa in, 372, 383
    treatment of, 407
  diet in, 390
  dyspepsia in, 371
    treatment of, 408
  dyspnœa in, 371
  emphysema and, 360, 365
  ETIOLOGY OF, chap. xxvii. 304 ff
  exercise in, 395, 396
  FIBROID, chap. xxxii. 342 ff.
    apyrexia in, 328 ff.
  fistula in ano, 372
  hæmic murmurs, absence of, 524
  heredity, 311, 312, 313
  hæmoptysis, prognosis of, 382; chap. xlvii. 521 ff.
  infection, 304 ff.
    food, 308, 309
    house, 306 ff.
    personal, 306
    congenital (cases), 309 ff.
    prevention of, 386 ff.
  infiltration (signs of), 373
  intestinal ulceration, 362
  kidneys in, 363, 383
  laryngeal, primary, 365, 366
    prognosis of, 382
  liver in, 362
  meningitis in, 363
  MILIARY, chap. xxx. 331 ff.
    pyrexia typical of, 325, 326
  nightsweats, treatment of, 406, 407
  onset, modes of, 364, 365, 366
  pain in, 369, 406
  PATHOLOGY OF, chap. xxvii 295 ff.
  peritonitis in, 362, 384
  pleural thickening, mode of production, 578, 579
  pleurisy and, 315; 545–550
  pneumonia in, 316, 383

Tuberculosis (*cont.*)
  pneumothorax in, 630 ff.
    arrest of process by, 632, 640
    'latent,' 633
    temperature in, 634, 635
    effusion in, 631
    prognosis in, 384, 640
    diagnosis from cavity, 639
    treatment of, 642
      (surgical), 643, 644
  pyopneumothorax in, surgical treatment of, 643, 644
  prognosis of, 379 ff.
  pulmonary vessels, thrombosis of, 518
    erosion of, 518
  pulse in, 371
  PYREXIA IN, TYPES OF, chap. xxix. 323 ff.
    inverse, 325 ff.
      importance in prognosis, 381, 382
    treatment of, 404
  sites and progress of lesions in, 350 ff.
  septic organisms in, 324, 589
  sex in, 314, 315
  sputa of, 366–369
  softening in, 375
  syphilis and, 317 and chap. xxxvii. 429 ff.
  tongue in, 371, 372
  TREATMENT OF (general), chap. xxxv. 385 ff.
    open air, 391 ff.
    medicinal, 401–4
    nucleins, 400
    serum, 400
    tuberculin, 397–9
  vomiting in, 372
Tubular breathing, 60
TUMOURS OF THE LUNGS (general), chap. xlii. 485 ff.
  surgical treatment of, 489 ff.
  pneumonic onset in, 488
TUMOURS OF PLEURA (general), chap. lx. 656 ff.
  effusion in, 662
    hæmorrhagic, 664
    characteristic cells in, 663
Turkish bath, value of, 109
'Turtle' lung, 125
Tympanitic resonance, 57
Typhus fever, bronchitis in, 115

VAGUS, pressure on, 155, 686, 693
Vasomotor system and pulmonary circulation, 277
'Veiled puff' (description, significance), 64, 135, 136
Venesection in capillary bronchitis, 99, 111
  in pneumonia, 233
Vesicular breathing, 58

WAVY breathing, 59, 374
Whooping cough, bronchitis in, 116
  pulmonary complications of, 533
Wounds of the lung, 494, 495

PRINTED BY
SPOTTISWOODE AND CO., NEW-STREET SQUARE
LONDON

A LIST OF WORKS ON MEDICINE, SURGERY AND GENERAL SCIENCE

CONTENTS

PAGE
ANATOMY 2
BACTERIOLOGY 17
BIOLOGY 12
CHEMISTRY 19
HEALTH AND HYGIENE 15
MEDICINE 2
MISCELLANEOUS 9
MONOGRAPHS ON BIOCHEMISTRY 24
OPTICS 18
PHOTOGRAPHY 18
PHYSIOLOGY 12
PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE ... 11
SURGERY 2
TEXT-BOOKS OF PHYSICAL CHEMISTRY 22
VETERINARY MEDICINE 11
ZOOLOGY 12

LONGMANS, GREEN, & CO.
39 PATERNOSTER ROW, LONDON, E.C.
91 AND 93 FIFTH AVENUE, NEW YORK
8 HORNBY ROAD, BOMBAY
303 BOWBAZAR STREET, CALCUTTA

MEDICINE, SURGERY, ANATOMY, ETC.

ASHBY AND WRIGHT. THE DISEASES OF CHILDREN, MEDICAL AND SURGICAL. By HENRY ASHBY, M.D. Lond., F.R.C.P., late Physician to the Manchester Children's Hospital; and G. A. WRIGHT, B.A., M.B. Oxon., F.R.C.S. Eng., Surgeon to the Manchester Royal Infirmary; Consulting Surgeon to the Manchester Children's Hospital. With 15 Plates (1 Coloured) and 241 Illustrations in the Text. Fifth Edition. Thoroughly Revised, 1905. 8vo, 21*s.* net.

BAIN AND EDGECOMBE. THE PHYSIOLOGY AND THERAPEUTICS OF THE HARROGATE WATERS, BATHS, AND CLIMATE APPLIED TO THE TREATMENT OF CHRONIC DISEASE. By WILLIAM BAIN, M.D., M.R.C.P., and WILFRID EDGECOMBE, M.D. 8vo, 7*s.* 6*d.* net.

BENNETT.—*WORKS by Sir WILLIAM H. BENNETT, K.C.V.O., F.R.C.S., Surgeon to St. George's Hospital.*

RECURRENT EFFUSION INTO THE KNEE-JOINT AFTER INJURY, WITH ESPECIAL REFERENCE TO INTERNAL DERANGEMENT, COMMONLY CALLED SLIPPED CARTILAGE: an Analysis of 750 Cases. A Clinical Lecture delivered at St. George's Hospital. With 13 Illustrations. 8vo, 3*s.* 6*d.*

CLINICAL LECTURES ON VARICOSE VEINS OF THE LOWER EXTREMITIES. With 3 Plates. 8vo, 6*s.*

ON VARICOCELE: A PRACTICAL TREATISE. With 4 Tables and a Diagram. 8vo, 5*s.*

CLINICAL LECTURES ON ABDOMINAL HERNIA: chiefly in relation to Treatment, including the Radical Cure. With 12 Diagrams in the Text. 8vo, 8*s.* 6*d.*

ON VARIX, ITS CAUSES AND TREATMENT, WITH ESPECIAL REFERENCE TO THROMBOSIS. 8vo, 3*s.* 6*d.*

LECTURE ON THE USE OF MASSAGE AND EARLY MOVEMENTS IN RECENT FRACTURES AND OTHER COMMON SURGICAL INJURIES: SPRAINS AND THEIR CONSEQUENCES: RIGIDITY OF THE SPINE, AND THE MANAGEMENT OF STIFF JOINTS GENERALLY. With 23 Illustrations. 8vo, 6*s.*

THE PRESENT POSITION OF THE TREATMENT OF SIMPLE FRACTURES OF THE LIMBS: an Address delivered to the British Medical Association. To which is appended a Summary of the Opinions and Practice of about 300 Surgeons. 8vo, 2*s.* 6*d.*

MEDICINE, SURGERY, ANATOMY, ETC.—*continued.*

CABOT. A GUIDE TO THE CLINICAL EXAMINATION OF THE BLOOD FOR DIAGNOSTIC PURPOSES. By RICHARD C. CABOT, M.D., Physician to Out-Patients, Massachusetts General Hospital. With 3 Coloured Plates and 28 Illus. in Text. 8vo, 16*s.*

COATS. A MANUAL OF PATHOLOGY. By JOSEPH COATS, M.D., late Professor of Pathology in the University of Glasgow. Fifth Edition, 1903. Revised throughout and Edited by LEWIS R. SUTHERLAND, M.D., Professor of Pathology, University of St. Andrews. With 729 Illustrations and 2 Coloured Plates. 8vo, 28*s.* net.

CHEYNE AND BURGHARD. A MANUAL OF SURGICAL TREATMENT. By Sir W. WATSON CHEYNE, Bart., C.B., M.B., F.R.C.S., F.R.S., D.Sc., Professor of Clinical Surgery in King's College, London; Surgeon to King's College Hospital, and the Children's Hospital, Paddington Green, etc.; and F. F. BURGHARD, M.D. and M.S. Lond., F.R.C.S., Teacher of Practical Surgery in King's College, London; Surgeon to King's College Hospital, and the Children's Hospital, Paddington Green, etc.

PART I. The treatment of General Surgical Diseases, including inflammation, suppuration, ulceration, gangrene, wounds and their complications, infective diseases and tumours; the administration of anæsthetics. With 66 Illustrations. Royal 8vo, 9*s.* net.

PART II. The treatment of the Surgical Affections of the Tissues, including the skin and subcutaneous tissues, the nails, the lymphatic vessels and glands, the fasciæ, bursæ, muscles, tendons and tendon-sheaths, nerves, arteries and veins; deformities. With 141 Illustrations. Royal 8vo, 12*s.* net.

PART III. The treatment of the Surgical Affections of the Bones. Amputations. With 100 Illustrations. Royal 8vo, 10*s.* 6*d.* net.

PART IV. The treatment of the Surgical Affections of the Joints (including excisions) and the spine. With 138 Illustrations. Royal 8vo, 12*s.* net.

PART V. The treatment of the Surgical Affections of the head, face, jaws, lips, larynx and trachea; and the Intrinsic Diseases of the nose, ear and larynx, by H. LAMBERT LACK, M.D. (Lond.), F.R.C.S., Surgeon to the Hospital for Diseases of the Throat, Golden Square, and to the Throat and Ear Department, the Children's Hospital, Paddington Green. With 145 Illustrations. Royal 8vo, 15*s.* net.

PART VI.—Section 1. The Surgical Affections of the tongue and floor of the mouth, the pharynx, neck, œsophagus, stomach and intestines. With an Appendix on the Examination of the Blood in Surgical Condition. By W. ESTE EMERY, M.D., D.Sc. (Lond.). With 124 Illustrations. Royal 8vo, 15*s.* net.

Section 2. The Surgical Affections of the rectum, the liver, pancreas and spleen, and genito-urinary organs, the breast and the thorax. With 113 Illustrations. Royal 8vo 18*s.* net.

MEDICINE, SURGERY, ANATOMY, ETC.—*continued.*

COOKE.—*WORKS by THOMAS COOKE, F.R.C.S. Eng., B.A., B.Sc., M.D. Paris, late Senior Assistant Surgeon to the Westminster Hospital.*

TABLETS OF ANATOMY. Being a Synopsis of demonstrations given in the Westminster Hospital Medical School. Eleventh Edition in three Parts, thoroughly brought up to date, and with over 700 Illustrations from all the best sources, British and Foreign. Post 4to. Part I. The Bones, 7*s.* 6*d.* net; Part II. Limbs, Abdomen, Pelvis, 10*s.* 6*d.* net; Part III. Head and Neck, Thorax, Brain, 10*s.* 6*d.* net.

APHORISMS IN APPLIED ANATOMY AND OPERATIVE SURGERY. Including 100 Typical *vivâ voce* Questions on Surface Marking, etc. Crown 8vo, 3*s.* 6*d.*

DAKIN. A HANDBOOK OF MIDWIFERY. By WILLIAM RADFORD DAKIN, M.D., F.R.C.P., Obstetric Physician and Lecturer on Midwifery at St. George's Hospital, Examiner in Midwifery and Diseases of Women on the Conjoint Board of the Royal Colleges of Physicians and Surgeons in England, etc. With 400 Illustrations. Large crown 8vo, 18*s.*

DICKSON. THE BONE-MARROW: a Cytological Study. Forming an Introduction to the Normal and Pathological Histology of the Tissue, more especially with regard to Blood Formatoin, Blood Destruction, etc. Together with a short account of the Reactions and Degenerations of the Tissue in Disease. By W. E. CARNEGIE DICKSON, M.D., B.Sc. Edin., F.R.C.P. Edin., Lecturer on Pathological Bacteriology and Senior Assistant to the Professor of Pathology in the University of Edinburgh; Assistant Pathologist to the Edinburgh Royal Infirmary. With 12 Coloured Plates and 51 Micro-Photographs by Richard Muir. Medium 4to, £2 2*s.* net.

DICKINSON.—*WORKS by W. HOWSHIP DICKINSON, M.D. Cantab., F.R.C.P., Consulting Physician to St. George's Hospital; Consulting Physician to the Hospital for Sick Children, etc.*

ON RENAL AND URINARY AFFECTIONS. Complete in Three Parts, 8vo, with 12 Plates, and 122 Woodcuts. £3 4*s.* 6*d.*

THE TONGUE AS AN INDICATION OF DISEASE; being the Lumleian Lectures delivered at the Royal College of Physicians in March, 1888. 8vo, 7*s.* 6*d.*

OCCASIONAL PAPERS ON MEDICAL SUBJECTS, 1855-1896. 8vo, 12*s.*

FOWLER AND GODLEE. THE DISEASES OF THE LUNGS. By JAMES KINGSTON FOWLER, M.A., M.D., F.R.C.P., Physician to the Middlesex Hospital and to the Hospital for Consumption and Diseases of the Chest, Brompton, etc.; and RICKMAN JOHN GODLEE, M.S., F.R.C.S., Honorary Surgeon-in-Ordinary to His Majesty, Fellow and Professor of Clinical Surgery, University College, London, etc. With 160 Illustrations. 8vo, 25*s.*

MEDICINE, SURGERY, ANATOMY, ETC.—*continued.*

GARROD. THE ESSENTIALS OF MATERIA MEDICA AND THERAPEUTICS. By Sir ALFRED BARING GARROD, M.D., F.R.S., etc.; late Vice-President of the Royal College of Physicians. Fourteenth Edition, Revised and Edited, under the Supervision of the Author, by NESTOR TIRARD, M.D. Lond., F.R.C.P., Professor of Materia Medica and Therapeutics in King's College, London, etc. Crown 8vo, 12*s.* 6*d.*

GOODSALL AND MILES. DISEASES OF THE ANUS AND RECTUM. By D. H. GOODSALL, F.R.C.S., late Senior Surgeon Metropolitan Hospital, Senior Surgeon St. Mark's Hospital; and W. ERNEST MILES, F.R.C.S., Assistant Surgeon to the Cancer Hospital, Surgeon (out-patients) to the Gordon Hospital, etc. (In Two Parts).

PART I.—Anatomy of the Ano-rectal Region—General Diagnosis—Abscess—Ano-rectal Fistula — Recto-urethral, Recto-vesical and Recto-vaginal Fistulæ—Sinus over the Sacro-coccygeal Articulation—Fissure—Hæmorrhoids (External and Internal). With 91 Illustrations. 8vo, 7*s.* 6*d.* net.

PART II.—Prolapse of the Rectum—Invagination of the Rectum—Ulceration—Stricture of the Anus and of the Rectum—Malignant Growths of the Anus and Rectum—Benign Tumours of the Anus and Rectum—Foreign Bodies in the Rectum—Pruritus Ani—Syphilis of the Anus and Rectum. With 44 Illustrations. 8vo, 6*s.* net.

GRAY. ANATOMY, DESCRIPTIVE AND APPLIED. By HENRY GRAY, F.R.S., Fellow of the Royal College of Surgeons, late Lecturer on Anatomy at St. George's Hospital Medical School. Seventeenth Edition. Edited by ROBERT HOWDEN, M.A., M.B., C.M., Professor of Anatomy in the University of Durham. Notes on Applied Anatomy, revised by A. J. JEX-BLAKE, M.A., M.B., M.R.C.P., Assistant Physician to St. George's Hospital; and W. FEDDE FEDDEN, M.S., F.R.C.S., Assistant Surgeon and Lecturer on Surgical Anatomy, St. George's Hospital. With 1,032 Illustrations. Royal 8vo, 32*s.* net.

HARE. THE FOOD FACTOR IN DISEASE: Being an investigation into the humoral causation, meaning, mechanism and rational treatment, preventive and curative, of the Paroxysmal Neuroses (migraine, asthma, angina pectoris, epilepsy, etc.), bilious attacks, gout, catarrhal and other affections, high blood-pressure, circulatory, renal and other degenerations. By FRANCIS HARE, M.D., late Consulting Physician to the Brisbane General Hospital; Visiting Physician at the Diamantina Hospital for Chronic Diseases, Brisbane; Inspector-General of Hospitals for Queensland. 2 vols. Medium 8vo, 30*s.* net.

MEDICINE, SURGERY, ANATOMY, ETC.—*continued.*

INFLUENCE OF HEREDITY ON DISEASE (THE), WITH SPECIAL REFERENCE TO TUBERCULOSIS, CANCER, AND DISEASES OF THE NERVOUS SYSTEM. A Discussion opened by SIR WILLIAM S. CHURCH, Bt., K.C.B., M.D., SIR WILLIAM R. GOWERS, M.D., F.R.S. (Diseases of the Nervous System), ARTHUR LATHAM, M.D. (Tuberculosis), and E. F. BASHFORD, M.D. (Cancer). [From the Proceedings of the Royal Society of Medicine, 1909, Vol. II., No. 3.] 4to, 4*s.* 6*d.* net.

LANG. THE METHODICAL EXAMINATION OF THE EYE. Being Part I. of a Guide to the Practice of Ophthalmology for Students and Practitioners. By WILLIAM LANG, F.R.C.S. Eng., Surgeon to the Royal London Ophthalmic Hospital, Moorfields, etc. With 15 illustrations. Crown 8vo, 3*s.* 6*d.*

LUFF. TEXT-BOOK OF FORENSIC MEDICINE AND TOXICOLOGY. By ARTHUR P. LUFF, M.D., B.Sc. Lond., Physician in Charge of Out-Patients and Lecturer on Medical Jurisprudence and Toxicology in St. Mary's Hospital; Examiner in Forensic Medicine in the University of London; External Examiner in Forensic Medicine in the Victoria University; Official Analyst to the Home Office. With 13 full-page Plates (1 in colours) and 33 Illustrations in the Text. 2 vols., Crown 8vo, 24*s.*

NEWMAN. MOVABLE KIDNEY AND OTHER DISPLACEMENTS AND MALPOSITIONS. By DAVID NEWMAN, M.D., F.F.P.S.G., Surgeon to the Glasgow Royal Infirmary. With 25 Illustrations. 8vo, 5*s.* net.

PROBYN-WILLIAMS. A PRACTICAL GUIDE TO THE ADMINISTRATION OF ANÆSTHETICS. By R. J. PROBYN-WILLIAMS, M.D., Anæsthetist and Instructor in Anæsthetics at the London Hospital; Lecturer in Anæsthetics at the London Hospital Medical College, etc. With 34 Illustrations. Crown 8vo, 4*s.* 6*d.* net.

MEDICINE, SURGERY, ANATOMY, ETC.—*continued.*

QUAIN. QUAIN'S (Sir RICHARD) DICTIONARY OF MEDICINE. By Various Writers. Edited by H. MONTAGUE MURRAY, M.D., F.R.C.P., Joint Lecturer on Medicine, Charing Cross Medical School, and Physician to Charing Cross Hospital, and to the Victoria Hospital for Children, Chelsea; Examiner in Medicine to the University of London. Assisted by JOHN HAROLD, M.B., B.Ch., B.A.O., Physician to St. John's and St. Elizabeth's Hospital, and Demonstrator of Medicine at Charing Cross Medical School, and W. CECIL BOSANQUET, M.A., M.D., F.R.C.P., Assistant Physician, Charing Cross Hospital, etc. Third and Cheaper Edition, largely Rewritten, and Revised throughout. With 21 Plates (14 in Colour) and numerous Illustrations in the Text. 8vo, 21*s*. net., buckram; 30*s*. net., half-morocco.

QUAIN. QUAIN'S (JONES) ELEMENTS OF ANATOMY. The Tenth Edition. Edited by EDWARD ALBERT SCHÄFER, F.R.S., Professor of Physiology in the University of Edinburgh; and GEORGE DANCER THANE, Professor of Anatomy in University College, London.

⁂ The several parts of this work form COMPLETE TEXT-BOOKS OF THEIR RESPECTIVE SUBJECTS. They can be obtained separately as follows:—

Vol. I., Part I. EMBRYOLOGY. By E. A. SCHÄFER, F.R.S. With 200 Illustrations. Royal 8vo, 9*s*.

Vol. I., Part II. GENERAL ANATOMY OR HISTOLOGY. By E. A. SCHÄFER, F.R.S. With 491 Illustrations. Royal 8vo, 12*s*. 6*d*.

Vol. II., Part I. OSTEOLOGY—ARTHROLOGY. By G. D. THANE. With 224 Illustrations. Royal 8vo, 11*s*.

Vol. II., Part II. MYOLOGY—ANGEIOLOGY. By G. D. THANE. With 199 Illustrations. Royal 8vo, 16*s*.

Vol. III., Part I. THE SPINAL CORD AND BRAIN. By E. A. SCHÄFER, F.R.S. With 139 Illustrations. Royal 8vo, 12*s*. 6*d*.

Vol. III., Part II. THE NERVES. By G. D. THANE. With 102 Illustrations. Royal 8vo, 9*s*.

Vol. III., Part III. THE ORGANS OF THE SENSES. By E. A. SCHÄFER, F.R.S. With 178 Illustrations. Royal 8vo, 9*s*.

Vol. III., Part IV. SPLANCHNOLOGY. By E. A. SCHÄFER, F.R.S., and JOHNSON SYMINGTON, M.D. With 337 Illustrations. Royal 8vo, 16*s*.

Appendix. SUPERFICIAL AND SURGICAL ANATOMY. By Professor G. D. THANE and Professor R. J. GODLEE, M.S. With 29 Illustrations. Royal 8vo, 6*s*. 6*d*.

MEDICINE, SURGERY, ANATOMY, ETC.—*continued.*

QUAIN. QUAIN'S ELEMENTS OF ANATOMY. The Eleventh Edition. Edited by EDWARD ALBERT SCHÄFER, F.R.S., Professor of Physiology and Histology in the University of Edinburgh; JOHNSON SYMINGTON, M.D., F.R.S., Professor of Anatomy in Queen's College, Belfast; and THOMAS HASTIE BRYCE, M.A., M.D., Regius Professor of Anatomy in the University of Glasgow.

IN FOUR VOLUMES. Royal 8vo.

VOL. I. EMBRYOLOGY. By T. H. BRYCE, M.A., M.D. Illustrated by more than 300 Engravings, many of which are coloured. 10*s.* 6*d.* net.

VOL. III. NEUROLOGY. By E. A. SCHÄFER and J. SYMINGTON.

Part I. Containing the General Structure of the Nervous System and the Structure of the Brain and Spinal Cord. With 361 Illustrations, many of which are coloured. 15*s.* net.

Part II. Containing the Descriptive Anatomy of the Peripheral Nerves and of the Organs of Special Sense. With numerous Illustrations, many of which are coloured. 15*s.* net.

⁂ *The other Volumes are in preparation.*

This work has been completely re-edited and brought up to date. The volumes will comprise respectively Embryology; General and Visceral Anatomy; the Nervous System and Sense Organs; and the Bones, Ligaments, Muscles, and Blood-vessels. Each volume will be complete in itself, and will serve as a text-book for the particular subject or subjects with which it deals. Thus the first volume is intended to form a complete text-book of Human Embryology, the second a text-book of Histology and Visceral Anatomy, the third a text-book of Neurology, the fourth dealing with the systems which are not included in the second and third volumes.

SCHÄFER. THE ESSENTIALS OF HISTOLOGY: Descriptive and Practical. For the Use of Students. By E. A. SCHÄFER, F.R.S., Professor of Physiology in the University of Edinburgh. With 553 Illustrations some of which are Coloured. Seventh Edition, 1907. 8vo, 10*s.* 6*d.* net.

SMALE AND COLYER. DISEASES AND INJURIES OF THE TEETH, including Pathology and Treatment. By MORTON SMALE, M.R.C.S., L.S.A., L.D.S., Dental Surgeon to St. Mary's Hospital, Consulting Dental Surgeon, Dental Hospital of London, etc.; and J. F. COLYER, L.R.C.P., M.R.C.S., L.D.S., Dental Surgeon to Charing Cross Hospital and to the Dental Hospital of London, Dean of the School, Dental Hospital of London. Second Edition Revised and Enlarged by J. F. COLYER. With 645 Illustrations. Large Crown 8vo, 21*s.* net.

MEDICINE, SURGERY, ANATOMY, ETC.—*continued.*

SMITH (H. F.). THE HANDBOOK FOR MIDWIVES. By HENRY FLY SMITH, B.A., M.B. Oxon., M.R.C.S. Second Edition. With 41 Woodcuts. Crown 8vo, 5*s.*

STEVENSON. WOUNDS IN WAR: the Mechanism of their Production and their Treatment. By Surgeon-General W. F. STEVENSON, C.B. (Army Medical Staff), B.A., M.B., M.Ch. Dublin University; Professor of Military Surgery, Royal Army Medical College, London. With 127 Illustrations. 8vo, 15*s.* net.

SYMINGTON. AN ATLAS OF SKIAGRAMS, ILLUSTRATING THE DEVELOPMENT OF THE TEETH. With Explanatory Text. By JOHNSON SYMINGTON, M.D., F.R.S., Professor of Anatomy, Queen's College, Belfast; and J. C. RANKIN, M.D., Physician in charge of the Electrical Department, Royal Victoria Hospital, Belfast. With 12 Plates. Demy 4to. 10*s.* 6*d.* net.

MISCELLANEOUS.

ANNUAL CHARITIES REGISTER AND DIGEST: being a Classified Register of Charities in or available for the Metropolis, together with a Digest of Information respecting the Legal, Voluntary, and other Means for the Prevention and Relief of Distress and the Improvement of the Condition of the Poor. With an elaborate Index, and an Introduction, "How to Help Cases of Distress". By C. S. LOCH, Secretary to the Council of the Charity Organisation Society, London. 8vo, 5*s.* net.

GASKELL. THE ORIGIN OF VERTEBRATES. By WALTER H. GASKELL, M.A., M.D. (Camb.), LL.D. (Edinburgh and McGill Univ., Montreal), F.R.S., Fellow of Trinity Hall and University Lecturer in Physiology, Cambridge. With 168 Illustrations. 8vo, 21*s.* net.

HOBART. THE MEDICAL LANGUAGE OF ST. LUKE. By the Rev. WILLIAM KIRK HOBART, LL.D. 8vo, 16*s.*

MISCELLANEOUS—*continued.*

HOPF. THE HUMAN SPECIES: CONSIDERED FROM THE STANDPOINTS OF COMPARATIVE ANATOMY, PHYSIOLOGY, PATHOLOGY AND BACTERIOLOGY. By Dr. LUDWIG HOPF. Authorised English Translation. With 7 Plates and 217 Illustrations in the Text. 8vo, 10*s.* 6*d.* net.

INQUIRY (AN) INTO THE PHENOMENA ATTENDING DEATH BY DROWNING AND THE MEANS OF PROMOTING RESUSCITATION IN THE APPARENTLY DROWNED. Report of a Committee appointed by the Royal Medical and Chirurgical Society. With 2 Diagrams and 26 Plates. 8vo, 5*s.* net.

KING'S COLLEGE HOSPITAL BOOK OF COOKING RECIPES: being a Collection of Recipes contributed by Friends of the Hospital and Published in aid of the Fund for the Removal of King's College Hospital to South London. Crown 8vo, 1*s.* net.

MOON. THE RELATION OF MEDICINE TO PHILOSOPHY. By R. O. MOON, M.A., M.D. (Oxon.), F.R.C.P., Physician to the National Hospital for Diseases of the Heart, etc. Crown 8vo.

PAGET. MEMOIRS AND LETTERS OF SIR JAMES PAGET, Bart., F.R.S., Sergeant-Surgeon to Her late Majesty Queen Victoria. Edited by STEPHEN PAGET, F.R.C.S. With Portrait. 8vo, 6*s.* net.

PETTIGREW. DESIGN IN NATURE: Illustrated by Spiral and other Arrangements in the Inorganic and Organic Kingdoms as exemplified in Matter, Force, Life, Growth, Rhythms, etc., especially in Crystals, Plants, and Animals. With Examples selected from the Reproductive, Alimentary, Respiratory, Circulatory, Nervous, Muscular, Osseous, Locomotory, and other Systems of Animals. By J. BELL PETTIGREW, M.D., LL.D., F.R.S., F.R.C.P.; Laureate of the Institute of France; late Chandos Professor of Anatomy and Medicine in the University, St. Andrews; Fellow of the Royal Botanical, Medico-Chirurgical, Royal Medical, Literary and Philosophical, Harveian and other Societies. Illustrated by nearly 2,000 Figures, largely original and from nature. In 3 vols. 4to. 63*s.* net.

POOLE. COOKERY FOR THE DIABETIC. By W. H. and Mrs. POOLE. With Preface by Dr. PAVY. Fcap. 8vo, 2*s.* 6*d.*

RAFFETY. AN INTRODUCTION TO THE SCIENCE OF RADIO-ACTIVITY. By CHARLES W. RAFFETY. With 27 Illustrations. Crown 8vo, 4*s.* 6*d.* net.

THE PROCEEDINGS OF THE ROYAL SOCIETY OF MEDICINE.

The Royal Society of Medicine was formed in June, 1907, by the amalgamation of the following London Medical Societies :—

The Royal Medical and Chirurgical Society.
The Pathological Society.
The Epidemiological Society.
The Odontological Society of Great Britain.
The Obstetrical Society.
The Clinical Society.
The Dermatological Society.
The British Gynæcological Society.
The Neurological Society.
The British Laryngological, Rhinological, and Otological Association.
The Laryngological Society.
The Dermatological Society of Great Britain and Ireland.
The Otological Society of the United Kingdom.
The British Electro-therapeutic Society.
The Therapeutical Society.

The "Proceedings" of the Royal Society of Medicine are published monthly from November to July inclusive. The numbers contain the papers of, and the discussions read at each of the Sections during the previous month, and are so arranged that each Section can, if desired, be detached and bound separately at the end of the year.

The Annual Subscription is **£3 3s. net,** *which may be paid through any bookseller.*

The price of each monthly number is **7s. 6d. net.**

VETERINARY MEDICINE, ETC.

FITZWYGRAM. HORSES AND STABLES. By Lieutenant-General Sir F. FITZWYGRAM, Bart. With 56 pages of Illustrations. 8vo, 3*s*. net.

HAYES. TRAINING AND HORSE MANAGEMENT IN INDIA. With Hindustanee Vocabulary. By M. HORACE HAYES, F.R.C.V.S. (late Captain, "The Buffs"). With Portrait. Crown 8vo, 8*s*. net.

STEEL.—*WORKS by JOHN HENRY STEEL, F.R.C.V.S., F.Z.S., A.V.D., late Professor of Veterinary Science and Principal of Bombay Veterinary College.*

A TREATISE ON THE DISEASES OF THE OX; being a Manual of Bovine Pathology. Especially adapted for the use of Veterinary Practitioners and Students. With 2 Plates and 117 Woodcuts. 8vo, 15*s*.

A TREATISE ON THE DISEASES OF THE SHEEP; being a Manual of Ovine Pathology for the use of Veterinary Practitioners and Students. With Coloured Plate and 99 Woodcuts. 8vo, 12*s*.

YOUATT.—*WORKS by WILLIAM YOUATT.*

THE HORSE. Revised and Enlarged by W. WATSON, M.R.C.V.S. With 52 Wood Engravings. 8vo, 7*s*. 6*d*.

THE DOG. Revised and Enlarged. With 33 Wood Engravings. 8vo, 6*s*.

PHYSIOLOGY, BIOLOGY, ZOOLOGY, ETC.

ASHBY. NOTES ON PHYSIOLOGY FOR THE USE OF STUDENTS PREPARING FOR EXAMINATION. By HENRY ASHBY, M.D. (Lond.), F.R.C.P., late Physician to the General Hospital for Sick Children, Manchester; Lecturer and Examiner in Diseases of Children in the Victoria University. With 148 Illustrations 18mo, 5*s.*

BARNETT. THE MAKING OF THE BODY: a Children's Book on Anatomy and Physiology. By Mrs. S. A. BARNETT. With 113 Illustrations. Crown 8vo, 1*s.* 9*d.*

BEDDARD. ELEMENTARY PRACTICAL ZOOLOGY. By FRANK E. BEDDARD, M.A. (Oxon.). With 93 Illustrations. Crown 8vo, 2*s.* 6*d.*

BIDGOOD. A COURSE OF PRACTICAL ELEMENTARY BIOLOGY. By JOHN BIDGOOD, B.Sc., F.L.S. With 226 Illustrations. Crown 8vo, 4*s.* 6*d.*

BOSE.—*WORKS by JAGADIS CHUNDER BOSE, M.A. (Cantab.), D.Sc. (Lond.), Professor, Presidency College, Calcutta.*

RESPONSE IN THE LIVING AND NON-LIVING. With 117 Illustrations. 8vo, 10*s.* 6*d.*

PLANT RESPONSE AS A MEANS OF PHYSIOLOGICAL INVESTIGATION. With 278 Illustrations. 8vo, 21*s.*

COMPARATIVE ELECTRO-PHYSIOLOGY: A PHYSICO PHYSIOLOGICAL STUDY. With 406 Illustrations and Classified List of 321 new Experiments. 8vo, 15*s.* net.

BRODIE. THE ESSENTIALS OF EXPERIMENTAL PHYSIOLOGY. For the use of Students. By T. G. BRODIE, M.D., Lecturer on Physiology, St. Thomas's Hospital Medical School. With 2 Plates and 177 Illustrations in the Text. Crown 8vo, 6*s.* 6*d.*

CHAPMAN. THE FORAMINIFERA: an Introduction to the Study of the Protozoa. By FREDERICK CHAPMAN, A.L.S., F.R.M.S. With 14 Plates and 42 Illustrations in the Text. 8vo, 9*s.* net.

PHYSIOLOGY, BIOLOGY, ZOOLOGY, ETC.—*continued.*

FURNEAUX. HUMAN PHYSIOLOGY. By W. FURNEAUX, F.R.G.S. With 223 Illustrations. Crown 8vo, 2*s.* 6*d.*

HALLIBURTON. THE ESSENTIALS OF CHEMICAL PHYSIOLOGY. For the Use of Students. By W. D. HALLIBURTON, M.D., F.R.S., F.R.C.P., Professor of Physiology in King's College, London. With 83 Illustrations. 8vo, 4*s.* 6*d.* net.

HUDSON AND GOSSE. THE ROTIFERA OR "WHEEL ANIMALCULES". By C. T. HUDSON, LL.D., and P. H. GOSSE, F.R.S. With 30 Coloured and 4 Uncoloured Plates. In 6 Parts. 4to, price 10*s.* 6*d.* each; Supplement, 12*s.* 6*d.* Complete in Two Volumes, with Supplement, 4to, £4 4*s.*

⁂ The Plates in the Supplement contain figures of almost all the Foreign Species, as well as of the British Species, that have been discovered since the original publication of Vols. I. and II.

LLOYD. THE TEACHING OF BIOLOGY IN THE SECONDARY SCHOOL. By FRANCIS E. LLOYD, A.M., and MAURICE A. BIGELOW, Ph.D., Professors in Teachers' College, Columbia University. Crown 8vo, 6*s.* net.

MOORE. ELEMENTARY PHYSIOLOGY AND ANATOMY. By BENJAMIN MOORE, D.Sc., Professor of Bio-Chemistry in the University of Liverpool. With 125 Illustrations. Crown 8vo, 3*s.* 6*d.*

MACALISTER.—*WORKS by ALEXANDER MACALISTER, M.D.*

AN INTRODUCTION TO THE SYSTEMATIC ZOOLOGY AND MORPHOLOGY OF VERTEBRATE ANIMALS. With 41 Diagrams. 8vo, 10*s.* 6*d.*

ZOOLOGY OF THE INVERTEBRATE ANIMALS. With 77 Diagrams. Fcp. 8vo, 1*s.* 6*d.*

ZOOLOGY OF THE VERTEBRATE ANIMALS. With 59 Diagrams. Fcp. 8vo, 1*s.* 6*d.*

MACDOUGALL.—*WORKS by DANIEL TREMBLY MACDOUGALL, Ph.D.*

TEXT-BOOK OF PLANT PHYSIOLOGY. With 159 Illustrations. 8vo, 7*s.* 6*d.* net.

ELEMENTARY PLANT PHYSIOLOGY. With 108 Illustrations. Crown 8vo, 3*s.*

PHYSIOLOGY, BIOLOGY, ZOOLOGY, ETC.—*continued.*

MORGAN. ANIMAL BIOLOGY. An Elementary Text-Book. By C. LLOYD MORGAN, F.R.S., Principal of University College, Bristol. With numerous Illustrations. Crown 8vo, 8*s.* 6*d.*

SCHÄFER. DIRECTIONS FOR CLASS WORK IN PRACTICAL PHYSIOLOGY: Elementary Physiology of Muscle and Nerve and of the Vascular and Nervous Systems. By E. A. SCHÄFER, LL.D., F.R.S., Professor of Physiology in the University of Edinburgh. With 48 Diagrams. 8vo, 3*s.* net.

THORNTON.—*WORKS by JOHN THORNTON, M.A.*

HUMAN PHYSIOLOGY. With 284 Illustrations, some of which are Coloured. Crown 8vo, 6*s.*

ELEMENTARY BIOLOGY, Descriptive and Experimental. With numerous Illustrations. Crown 8vo, 3*s.* 6*d.*

ELEMENTARY PRACTICAL PHYSIOLOGY. With 178 Illustrations (6 of which are Coloured). Crown 8vo, 3*s.* 6*d.*

WALLER.—*WORKS by AUGUSTUS D. WALLER, M.D., F.R.S., Hon. LL.D. Edin., Lecturer on Physiology at St. Mary's Hospital Medical School, London; late External Examiner at the Victorian University.*

AN INTRODUCTION TO HUMAN PHYSIOLOGY. With 314 Illustrations. 8vo, 18*s.*

LECTURES ON PHYSIOLOGY. First Series.—*On Animal Electricity.* 8vo, 5*s.* net.

HEALTH AND HYGIENE, ETC.

ASHBY. HEALTH IN THE NURSERY. By HENRY ASHBY, M.D., F.R.C.P., Physician to the General Hospital for Sick Children, Manchester; Lecturer and Examiner in Diseases of Children in the Victoria University. With 25 Illustrations. Crown 8vo, 3*s*. net.

BUCKTON. HEALTH IN THE HOUSE. By Mrs. C. M. BUCKTON. With 41 Woodcuts and Diagrams. Crown 8vo, 2s.

BULL.—*WORKS by THOMAS BULL, M.D. Thoroughly Revised by ROBERT W. PARKER, M.R.C.S. Eng.*

HINTS TO MOTHERS ON THE MANAGEMENT OF THEIR HEALTH DURING THE PERIOD OF PREGNANCY, AND HINTS ON NURSING. Fcp. 8vo, sewed, 1*s*. 6*d*.; cloth, gilt edges, 2*s*. net.

THE MATERNAL MANAGEMENT OF CHILDREN IN HEALTH AND DISEASE. Fcp. 8vo, sewed, 1*s*. 6*d*.; cloth, gilt edges, 2*s*. net.

BUTTERWORTH. MANUAL OF HOUSEHOLD WORK AND MANAGEMENT. By ANNIE BUTTERWORTH. Crown 8vo, 2*s*. 6*d*.

CORFIELD. THE LAWS OF HEALTH. By W. H. CORFIELD, M.A., M.D. Fcp. 8vo, 1*s*. 6*d*.

CREIGHTON. THE ECONOMICS OF THE HOUSEHOLD. Six Lectures given at the London School of Economics during the Winter of 1906. By LOUISE CREIGHTON. Crown 8vo, 1*s*. 4*d*.

FURNEAUX. ELEMENTARY PRACTICAL HYGIENE. Section I. By WILLIAM S. FURNEAUX. With 146 Illustrations. Crown 8vo 2*s*. 6*d*.

JAMES. BALL GAMES AND BREATHING EXERCISES. By ALICE R. JAMES. With Preface by HARRY CAMPBELL, M.D., B.S. (London), F.R.C.P. With 17 Illustrations. Crown 8vo, 1*s*. 6*d*.

HEALTH AND HYGIENE, ETC.—*continued.*

NOTTER AND FIRTH.—*WORKS by J. LANE NOTTER, M.A., M.D., and R. H. FIRTH, F.R.C.S.*

HYGIENE. With 99 Illustrations. Crown 8vo, 4*s.* 6*d.*

PRACTICAL DOMESTIC HYGIENE. With 84 Illustrations. Crown 8vo, 2*s.* 6*d.*

POORE.—*WORKS by GEORGE VIVIAN POORE, M.D., F.R.C.P.*

THE EARTH IN RELATION TO THE PRESERVATION AND DESTRUCTION OF CONTAGIA: being the Milroy Lectures delivered at the Royal College of Physicians in 1899, together with other Papers on Sanitation. 13 Illustrations. Crown 8vo, 5*s.*

ESSAYS ON RURAL HYGIENE. With 12 Illustrations. Crown 8vo, 6*s.* 6*d.*

THE DWELLING HOUSE. With 36 Illustrations. Crown 8vo, 3*s.* 6*d.*

COLONIAL AND CAMP SANITATION. With 11 Illustrations. Crown 8vo, 2*s.* net.

PORTER. SCHOOL HYGIENE AND THE LAWS OF HEALTH: a Text-Book for Teachers and Students in Training. By CHARLES PORTER, M.D., B.Sc., M.R.C.P. (Edin.). With 121 Illustrations. Crown 8vo, 3*s.* 6*d.*

ROBINSON. THE HEALTH OF OUR CHILDREN IN THE COLONIES: a Book for Mothers. By LILIAN AUSTEN ROBINSON, M.D. Crown 8vo, 2*s.* 6*d.* net.

TUCKER. NOTES ON THE CARE OF BABIES AND YOUNG CHILDREN. For the Use of Teachers. By BLANCHE TUCKER, Inspectress of Elementary Schools. With an Introduction by DR. HOPE, Medical Officer of Health for Liverpool. Crown 8vo, 1*s.*

WEST. HOW TO NURSE SICK CHILDREN. By CHARLES WEST, M.D., Founder of and late Physician to the Hospital for Sick Children, Great Ormond Street, London. With Preface by GEORGE F. STILL, M.D., Physician to the Hospital for Sick Children, Great Ormond Street. Crown 8vo, 1*s.* net.

BACTERIOLOGY, ETC.

CURTIS. THE ESSENTIALS OF PRACTICAL BACTERIOLOGY: **an Elementary Laboratory Work for Students and Practitioners. By H. J. CURTIS, B.S. and M.D. Lond., F.R.C.S., formerly Surgeon to the North-Eastern Hospital for Children; Assistant Surgeon, Royal Hospital for Children and Women, Waterloo Road; Surgical Registrar and Assistant to the Professor of Pathology, University College, London. With 133 Illustrations. 8vo, 9*s*.**

ELLIS. OUTLINES OF BACTERIOLOGY (Technical and Agricultural). By DAVID ELLIS, Ph.D. (Marburg), D.Sc. (London), F.R.S.E., Lecturer in Bacteriology and Botany to the Glasgow and West of Scotland Technical College, Glasgow. With Illustrations. 8vo, 7*s*. 6*d*. net.

FRANKLAND. BACTERIA IN DAILY LIFE. By Mrs. PERCY FRANKLAND, F.R.M.S. Crown 8vo, 5*s*. net.

GOADBY. THE MYCOLOGY OF THE MOUTH: A TEXT-BOOK OF ORAL BACTERIA. By KENNETH W. GOADBY, L.D.S. Eng., D.P.H. Camb., L.R.C.P., M.R.C.S., Bacteriologist and Lecturer on Bacteriology, National Dental Hospital, etc. With 82 Illustrations. 8vo, 8*s*. 6*d*. net.

KLÖCKER. FERMENTATION ORGANISMS. A Laboratory Handbook. By ALB. KLÖCKER, Assistant in the Carlsberg Laboratory, Copenhagen. Translated from the German by G. E. ALLAN, B.Sc., Lecturer in the University of Birmingham, and J. H. MILLAR, F.I.C., formerly Lecturer in the British School of Malting and Brewing, and revised by the Author. With 146 Illustrations. 8vo, 12*s*. net.

OPTICS, PHOTOGRAPHY, ETC.

ABNEY. A TREATISE ON PHOTOGRAPHY. By Sir WILLIAM DE WIVELESLIE ABNEY, K.C.B., F.R.S. With 134 Illustrations. Crown 8vo, 5*s.*

BALY.—SPECTROSCOPY. By E. C. C. BALY, F.I.C., Lecturer on Spectroscopy and Assistant Professor of Chemistry, University College, London. With 163 Illustrations. Crown 8vo, 10*s.* 6*d.*

DRUDE. THE THEORY OF OPTICS. By PAUL DRUDE, Professor of Physics at the University of Giessen. Translated from the German by C. RIBORG MANN and ROBERT A. MILLIKAN, Professors of Physics at the University of Chicago. With 110 Diagrams. 8vo, 15*s.* net.

GLAZEBROOK. PHYSICAL OPTICS. By R. T. GLAZEBROOK, M.A., F.R.S. With 183 Woodcuts of Apparatus, etc. Crown 8vo, 6*s.*

POLLOK. PRACTICAL SPECTROGRAPHIC ANALYSIS. By J. H. POLLOK, D.Sc. Crown 8vo.

SHEPPARD AND MEES. INVESTIGATION ON THE THEORY OF THE PHOTOGRAPHIC PROCESS. By S. E. SHEPPARD, D.Sc. (Lond.), 1851 Exhibition Scholar of University College, London, and C. E. KENNETH MEES, D.Sc. (Lond.). With 65 Illustrations and Diagrams. Crown 8vo, 6*s.* 6*d.* net.

VANDERPOEL. COLOUR PROBLEMS: A Practical Manual for the Lay Student of Colour. By EMILY NOYES VANDERPOEL. With 117 Plates in Colour. Square 8vo, 21*s.* net.

WRIGHT. OPTICAL PROJECTION: A Treatise on the Use of the Lantern in Exhibition and Scientific Demonstration. By LEWIS WRIGHT, Author of "Light: a Course of Experimental Optics". With 232 Illustrations. Crown 8vo, 6*s.*

CHEMISTRY, ETC.

ARMITAGE. A HISTORY OF CHEMISTRY.
By F. P. ARMITAGE, M.A., F.C.S. Crown 8vo, 6s.

ARRHENIUS.—*WORKS by SVANTE ARRHENIUS, Director of the Nobel Institute, Stockholm.*

THEORIES OF CHEMISTRY: being Lectures delivered at the University of California, in Berkeley. Edited by T. SLATER PRICE, D.Sc., Ph.D., F.I.C. 8vo, 5s. 6d. net.

A TEXT-BOOK OF ELECTRO-CHEMISTRY. Translated from the German Edition by JOHN McCRAE, Ph.D. With 58 Illustrations. 8vo, 9s. 6*d*. net.

BUNGE. TEXT-BOOK OF ORGANIC CHEMISTRY FOR MEDICAL STUDENTS. By Dr. G. VON BUNGE, Professor of Physiology in the University of Basel. Translated by R. H. ADERS PLIMMER, D.Sc. 8vo, 6*s*. net.

CROOKES. SELECT METHODS IN CHEMICAL ANALYSIS (chiefly inorganic). By Sir W. CROOKES, F.R.S. With 68 Illustrations. 8vo, 21*s*. net.

FINDLAY.—*WORKS by ALEX. FINDLAY, M.A., Ph.D., D.Sc.*

PHYSICAL CHEMISTRY AND ITS APPLICATIONS IN MEDICAL AND BIOLOGICAL SCIENCE. Being a Course of Seven Lectures delivered in the University of Birmingham. Royal 8vo, 2*s*. net.

PRACTICAL PHYSICAL CHEMISTRY With 92 Illustrations. Crown 8vo, 4*s*. 6*d*.

HANSON AND DODGSON. AN INTERMEDIATE COURSE OF LABORATORY WORK IN CHEMISTRY. By EDWARD KENNETH HANSON, M.A. (Cant.), F.I.C., Teachers' Diploma (Lond.); Lecturer in Chemistry, University College, Reading; Lecturer to the Cambridge University Local Lecture Syndicate, and JOHN WALLIS DODGSON, B.Sc. (Lond.); Director of Evening Classes and Lecturer in Chemistry, University College, Reading. With Illustrations. 8vo, 3*s*. 6*d*.

MENDELEEFF. THE PRINCIPLES OF CHEMISTRY. By D. MENDELÉEFF. Translated from the Russian (Seventh Edition) by GEORGE KAMENSKY, A.R.S.M., and Edited by THOMAS H. POPE, B.Sc., F.I.C. With 110 Illustrations. 2 vols. 8vo, 32*s*. net.

MEYER. OUTLINES OF THEORETICAL CHEMISTRY. By LOTHAR MEYER. Translated by Professors P. PHILLIPS BEDSON, D.Sc., and W. CARLETON WILLIAMS, B.Sc. 8vo, 9*s*.

MUIR. A COURSE OF PRACTICAL CHEMISTRY.
By M. M. P. MUIR, M.A.

Part I. Elementary. Cr. 8vo, 4*s*. 6*d*. Part II. Intermediate. Cr. 8vo, 4*s*. 6*d*.

CHEMISTRY, ETC.—*continued.*

NEWTH.—*WORKS by G. S. NEWTH, F.I.C., F.C.S.*

CHEMICAL LECTURE EXPERIMENTS. With 230 Illustrations. Crown 8vo, 6*s.*

CHEMICAL ANALYSIS, QUANTITATIVE AND QUALITATIVE. With 102 Illustrations. Crown 8vo, 6*s.* 6*d.*

SMALLER CHEMICAL ANALYSIS. Crown 8vo, 2*s.*

A TEXT-BOOK OF INORGANIC CHEMISTRY. With 15 Illustrations. Crown 8vo, 6*s.* 6*d.*

ELEMENTARY PRACTICAL CHEMISTRY. With 108 Illustrations and 254 Experiments. Crown 8vo, 2*s.* 6*d.*

PERKIN.—*WORKS by F. MOLLWO PERKIN, Ph.D.*

QUALITATIVE CHEMICAL ANALYSIS (ORGANIC AND INORGANIC). With 15 Illustrations and Spectrum Plate. 8vo, 4*s.*

PRACTICAL METHODS OF ELECTRO-CHEMISTRY. 8vo, 6*s.* net.

PRICE AND TWISS. A COURSE OF PRACTICAL ORGANIC CHEMISTRY. By T. SLATER PRICE, D.Sc., Ph.D., F.I.C., Head of the Chemical Department of the Birmingham Municipal Technical School, and D. F. TWISS, M.Sc., A.I.C., Lecturer in Chemistry at the Birmingham Municipal Technical School. 8vo, 3*s.* 6*d.*

RADCLIFFE AND SINNATT. A SYSTEMATIC COURSE OF PRACTICAL ORGANIC CHEMISTRY. By LIONEL GUY RADCLIFFE, F.C.S. With the assistance of FRANK STURDY SINNATT, F.C.S. 8vo, 4*s.* 6*d.*

REYNOLDS. EXPERIMENTAL CHEMISTRY for Junior Students. By J. EMERSON REYNOLDS, M.D., F.R.S. Fcap. 8vo, with numerous Illustrations.

PART I.—*Introductory*, 1*s.* 6*d.* PART III.—*Metals and Allied Bodies*, 3*s.* 6*d.*

PART II.—*Non-Metals*, 2*s.* 6*d.* PART IV.—*Chemistry of Carbon Compounds*, 4*s.*

SMITH AND HALL. THE TEACHING OF CHEMISTRY AND PHYSICS IN THE SECONDARY SCHOOL. By ALEXANDER SMITH, B.Sc., Ph.D., Associate Professor of Chemistry in the University of Chicago, and EDWIN H. HALL, Ph.D., Professor of Physics in Harvard University. With 21 Woodcuts. Bibliographies, and Index. Crown 8vo, 6*s.* net.

CHEMISTRY, ETC.—*continued.*

STEWART. RECENT ADVANCES IN ORGANIC CHEMISTRY. By A. W. STEWART, D.Sc., Lecturer on Stereochemistry in University College, London. With an Introduction by J. NORMAN COLLIE, Ph.D., LL.D., F.R.S., Professor of Organic Chemistry in University College, London. 8vo, 7*s.* 6*d.*

THORPE. A DICTIONARY OF APPLIED CHEMISTRY. By Sir T. E. THORPE, C.B., D.Sc. Vict., Ph.D., F.R.S., Principal o Government Laboratory, London. Assisted by Eminent Contributors 3 vols. 8vo. Vols. I. and II., £2 2*s.* each; Vol. III., £3 3*s.*

TILDEN.—*Works by WILLIAM A. TILDEN, D.Sc. London, F.R.S.*

A SHORT HISTORY OF THE PROGRESS OF SCIENTIFIC CHEMISTRY IN OUR OWN TIMES. Crown 8vo, 5*s.* net.

INTRODUCTION TO THE STUDY OF CHEMICAL PHILOSOPHY. The Principles of Theoretical and Systematic Chemistry. With 5 Illustrations. Crown 8vo, 5*s.* With ANSWERS to Problems Crown 8vo, 5*s.* 6*d.*

PRACTICAL CHEMISTRY. The Principles of Qualitative Analysis Fcp. 8vo, 1*s.* 6*d.*

WATTS' DICTIONARY OF CHEMISTRY. Revised and entirely Re-written by H. FORSTER MORLEY, M.A., D.Sc., Fellow of, and lately Assistant-Professor of Chemistry in, University College, London and M. M. PATTISON MUIR, M.A., F.R.S.E., Fellow and Prælector in Chemistry of Gonville and Caius College, Cambridge. Assisted by Eminent Contributors. 4 vols. 8vo, £5 net.

WESTON. A SCHEME FOR THE DETECTION OF THE MORE COMMON CLASSES OF CARBON COMPOUNDS By FRANK E. WESTON, B.Sc., London (First Class Honours), F.C.S. Lecturer in Chemistry at the Polytechnic, Regent Street, W. 8vo, 2*s.*

WHITELEY.—*WORKS by R. L. Whiteley, F.I.C., Principal of the Municipal Science School, West Bromwich.*

CHEMICAL CALCULATIONS. With Explanatory Notes, Problems and Answers, specially adapted for use in Colleges and Science Schools With a Preface by Professor F. CLOWES, D.Sc. (Lond.), F.I.C. Crown 8vo, 2*s.*

ORGANIC CHEMISTRY: the Fatty Compounds. With 45 Illustrations. Crown 8vo, 3*s.* 6*d.*

TEXT-BOOKS OF PHYSICAL CHEMISTRY.

Edited by Sir WILLIAM RAMSAY, K.C.B., F.R.S., D.Sc.

STOICHIOMETRY. By SYDNEY YOUNG, D.Sc., F.R.S., Professor of Chemistry in the University of Dublin; together with AN INTRODUCTION TO THE STUDY OF PHYSICAL CHEMISTRY, by SIR WILLIAM RAMSAY, K.C.B., F.R.S., Editor of the Series. Crown 8vo 7*s*. 6*d*.

AN INTRODUCTION TO THE STUDY OF PHYSICAL CHEMISTRY. Being a General Introduction to the Series by SIR WILLIAM RAMSAY, K.C.B., F.R.S., D.Sc. Crown 8vo, 1*s*. net.

CHEMICAL STATICS AND DYNAMICS, INCLUDING THE THEORIES OF CHEMICAL CHANGE, CATALYSIS, AND EXPLOSIONS. By J. W. MELLOR, D.Sc. (N.Z.), B.Sc. (Vict.). Crown 8vo, 7*s*. 6*d*.

THE PHASE RULE AND ITS APPLICATIONS. By ALEX. FINDLAY, M.A., Ph.D., D.Sc., Lecturer and Demonstrator in Chemistry, University of Birmingham. With 134 Figures in the Text. Crown 8vo, 5*s*.

SPECTROSCOPY. By E. C. C. BALY, F.I.C., Lecturer on Spectroscopy and Assistant Professor of Chemistry, University College, London. With 163 Illustrations. Crown 8vo, 10*s*. 6*d*.

THERMOCHEMISTRY. By JULIUS THOMSEN, Emeritus Professor of Chemistry in the University of Copenhagen. Translated by KATHARINE A. BURKE, B.Sc. (Lond.), Assistant in the Department of Chemistry, University College, London. Crown 8vo, 9*s*.

TEXT-BOOKS OF PHYSICAL CHEMISTRY, E

—continued.

ELECTRO-CHEMISTRY. PART I.—GENERAL TH
By R. A. LEHFELDT, D.Sc., Professor of Physics at the East
Technical College. Including a Chapter on the Relation of Chemi
stitution to Conductivity, by T. S. MOORE, B.A., B.Sc., Lec
the University of Birmingham. Crown 8vo, 5*s.*

ELECTRO-CHEMISTRY. PART II.—APPLICATION
ELECTROLYSIS, PRIMARY AND SECONDARY
TERIES, ETC. By N. T. M. WILSMORE, D.Sc. Crown
[*In pre*

STEREOCHEMISTRY. By A. W. STEWART, D.Sc., Carnegie
Fellow, Lecturer on Stereochemistry in University College, Londo
87 Illustrations. Crown 8vo, 10*s.* 6*d.*

THE THEORY OF VALENCY. By J. NEWTON FRIEN
(Würz), M.Sc. (Birmingham). Crown 8vo, 5*s.*

RELATIONS BETWEEN CHEMICAL CONSTITUTIO
PHYSICAL PROPERTIES. By SAMUEL SMILE
Crown 8vo. [*In pre*

THERMODYNAMICS. By F. G. DONNAN, M.A., Ph.D. Crown
[*In pre*

ACTINOCHEMISTRY. By C. E. K. MEES, D.Sc., and S. E.
PARD, D.Sc. Crown 8vo. [*In pre*

PRACTICAL SPECTROGRAPHIC ANALYSIS. By J. H. P
D.Sc. Crown 8vo. [*In pre*

MONOGRAPHS ON BIOCHEMISTRY.

Edited by R. H. ADERS PLIMMER, D.Sc., and F. GOWLAND HOPKINS, D.Sc., F.R.S.

Royal 8vo.

In these volumes an attempt is being made to make the subject of Biochemistry more accessible by issuing a series of monographs upon the various chapters of the subject, each independent of and yet dependent upon the others, so that from time to time, as new material and the demand therefor necessitate, a new edition of each monograph can be issued without reissuing the whole series. The expenses of publication and the expense to the purchaser will thus be diminished, and by a moderate outlay it will be possible to obtain a full account of any particular subject as nearly current as possible.

THE DEVELOPMENT AND PRESENT POSITION OF BIOLOGICAL CHEMISTRY. By F. GOWLAND HOPKINS, M.A., M.B., D.Sc., F.R.S. [*In preparation.*

THE NATURE OF ENZYME ACTION. By W. M. BAYLISS, D.Sc., F.R.S. 3*s.* net.

THE CHEMICAL CONSTITUTION OF THE PROTEINS. By R. H. ADERS PLIMMER, D.Sc. In 2 Parts. Part 1, 3*s.* net; Part 2, 2*s.* 6*d.* net.

THE GENERAL CHARACTERS OF THE PROTEINS. By S. B. SCHRYVER, D.Sc., Ph.D. 2*s.* 6*d.* net.

THE VEGETABLE PROTEINS. By THOMAS B. OSBORNE, Ph.D. 3*s.* 6*d.* net.

THE POLYSACCHARIDES. By ARTHUR R. LING, F.I.C. [*In preparation.*

GLUCOSE AND THE GLUCOSIDES. By E. FRANKLAND ARMSTRONG, D.Sc., Ph.D. [*In preparation.*

THE FATS. By J. B. LEATHES, D.Sc. [*In preparation.*

COLLOIDS. By W. B. HARDY, M.A., F.R.S. [*In preparation.*

H.—2,500—A. U. P.—ix/1909.

L756 Fowler, James K.
F786 Diseases of the
1898 lungs 28801

NAME	DATE DUE
Gerlsen SN	MAR 14 1966